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The Bradshaw Lecture

ON

THE RESULTS OF BRONCHIAL OBSTRUCTION.

DELIVERED BEFORE THE ROYAL COLLEGE OF PHYSICIANS OF LONDON ON NOVEMBER 1st, 1910.

BY

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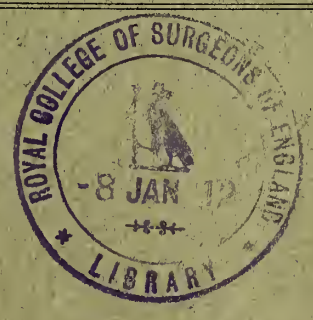

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THE RESULTS OF BRONCHIAL OBSTRUCTION.

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By G. NEWTON PITT, M.D.Cantab., F.R.C.P.Lond,
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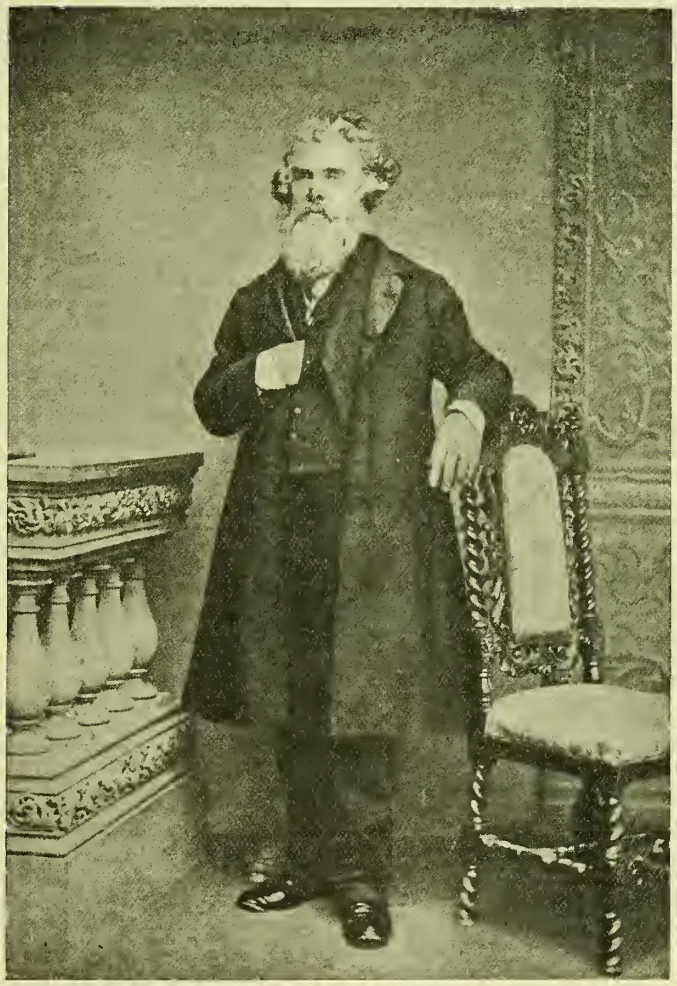
Mr. President and Gentlemen,—My first duty is to thank your predecessor in the chair for the honour he has done me in nominating me for this lectureship to-day, and to express the diffidence I feel in choosing a subject on which it may seem there is not much that is new to be said.

We are met to-day to do honour to the memory of Dr. William Wood Bradshaw, who, it would appear, was a man of wide interests—he contributed papers on such subjects as the use of cod-liver oil in chronic rheumatism, on narcotics, on abdominal abscess, and other non-professional articles to the *Miscellany* and elsewhere. Born in 1801 at Bristol, according both to Foster's *Alumni Oxoniensis* and to the inscription on his grave, he was educated at the Westminster and Middlesex Hospitals, taking his M.R.C.S. in 1833. In the early part of the same year he became M.D.Erlangen. He became an Extra Licentiate of this College in 1841, after examination by the President and the "Elects," which entitled him to practise beyond seven miles from London. In 1854 he was elected a Fellow of the Royal College of Surgeons of England. In 1859 Dr. Coplans Hawkins and Dr. R. P. Smith nominated him as a Member of this College, which nomination was approved. At the mature age of 43 he matriculated at Newton Hall, Oxford, and was granted a degree in 1847 without any examination. He married the widow of a wealthy jeweller at Andover, and, according to Mr. Rickman Godlee, to whom I am indebted for his portrait and for much of this information, her money was to be left away in case she remarried; but they bought the reversion, married, and settled down at Reading. He seems to have been a cultivated, refined, somewhat eccentric man with a rather theatrical manner, and never did much practice. He died in 1866; his widow founded a lectureship at each of the Colleges in order to perpetuate his memory.



Dr. William Wood Bradshaw.

The Results of Compression of a Bronchus.

The results of compression of a bronchus may be the following:

1. The lung may be slightly reduced in size. There may be definite diminution in the entry of air, but the resonance may be unaltered, and even *post mortem* beyond an increase in the amount of mucoid contents, and a diminution in the amount of air in the lung, this may be all that can be observed, even when there has been definite pressure on the tube.

2. The chest may be hyper-resonant, the lung over-distended, and the diaphragm displaced down abnormally. This occurs only with partial obstruction of the tube when respiration is vigorous, and will be discussed under the results produced by aneurysm.

3. The lung may be reduced in size, airless and solid from collapse. The chest will be dull on percussion. This results when the obstruction to the tube is complete, or when respiration is feeble. It may occur within a few hours, and was studied by Lichtheim by observing the effects of introducing foreign bodies into the bronchi so as to completely block them, but practically complete obstruction is generally due to other causes, and rarely to the presence of a foreign body. In the early stage the bronchi beyond the obstruction are empty. The lungs in the first and second conditions also tend gradually to become more or less airless.

4. The bronchi become filled with retained secretions, at first chiefly mucus, but catarrhal changes soon ensue and cells accumulate, with purulent and inflammatory products as well.

5. Secondary inflammatory changes take place, due partly to the entry of secretions into the alveoli, so that what has been called a retention pneumonia ensues; the alveoli fill with catarrhal cells and giant cells appear, although they are unconnected with tubercle, as has been shown by inoculating animals with the products. When alveolar walls remain long in contact, organization with obliteration takes place and new connective tissue is formed. The bronchi normally often contain a few staphylococci and streptococci, which when retained in suitable media begin to develop, with the result that pyrexia and a more or less acute pneumonic change develops.

The changes that have taken place so far may all pass away and leave but little damage; but two further changes soon take place—a general fibrosis of the lung beyond the obstruction and a bronchiectasis. These ultimately develop, whatever be the initial process, if the obstruction persist.

6. Dilatation of the tubes. This does not take place equally in all the tubes obstructed, and its distribution is largely determined by local causes. When only one branch of a bronchus has been pressed upon, the dilatation is limited to the tubes in its area. Generally the tubes at the posterior and lower part of the lung are the most affected, probably because the secretion tends to stagnate there, and set up local destruc-

tive changes, as the patient is in the semirecumbent position.

Normally there is a constant secretion of mucus in the bronchi, which passes imperceptibly out into the larger tubes and thence to the trachea, but we are quite ignorant what is the amount normally secreted in twenty-four hours. When a bronchus is occluded the mucus at once begins to accumulate, and is capable of being secreted under pressure, so that as with an obstructed gall bladder the tubes become over-distended. Now bronchial contents cannot pass through the bronchial wall on account of the basement membrane; they can only pass out by the bronchus, or be sucked back by inspiration into the alveoli which remain patent, and so on into the lymphatics and the glands. Normally the contents pass almost entirely by the bronchus, because the initial act of expiration consists in the contraction of the lung by its own elasticity, which as the alveoli empty drives the secretion out of the smaller bronchioles into the larger tubes, to be then carried on by the forcible muscular expiration. With a collapsed lung this initial mechanism fails. As the alveoli become blocked, there is a greater suction on those that remain, and this leads to further occlusion of the alveoli, but the movement of the chest on the affected side is always more or less feeble. The result is that, whether the lung is distended or collapsed, the bronchi become distended with mucoid or muco-purulent contents under high pressure, and hence the walls yield. Owing to the interference with the circulation, which is largely dependent on the suction of the alveoli as they open up with inspiration, the arteries being on the proximal and the veins on the distal side, the tissues are insufficiently nourished, and this, added to the local irritation, causes the bronchial walls to deteriorate, so that the elastic and muscle fibres tend to disappear and local dilatations occur.

It is an old controversy as to whether inspiratory or expiratory efforts are the more efficient in the production of bronchiectases, and I am sorry to say that at the present day the latter theory seems to have the more advocates. In the cases under consideration it is difficult to see how violent expiration, compressing the lung forcibly, as with a cough, can throw any greater strain on the inside of the bronchi than it does on the outside. If the same pressure is applied all round the wall of a bronchus it cannot tend to dilate it. The increased pressure of the contained air is the result of the pressure applied to the outer surface of the bronchus. On the other hand, if it is borne in mind that aspiration is applied to the surface of the lung by inspiration, which tends to open it up, and if the alveoli fail to respond, strain is thrown on the deeper parts, such as the bronchi; if there is no obstruction, they open up and fill with air from the trachea, but when the bronchus is obstructed they must be subjected to strain which tends to dilate them. The feebleness of the respiratory movements spares them greatly, but the contracted condition of the lung tends to set up an inspiratory dyspnoea, and paroxysms occur from time to time, especially with exertion, which must aspirate the tubes and gradually make them dilate.

Consequently we do not find marked dilatation except when there is also in these cases definite fibrosis. The slighter degrees of dilatation are associated with but slight consolidation of the lung. The presence of pleuritic effusion usually inhibits, or entirely prevents, the dilatation of the bronchi, because the lung, not being in contact with the pleura, is no longer held up by capillary attraction between the two surfaces, and therefore is not subjected to so much aspiration. There is no evidence that connective tissue in contracting is capable of producing traction on the tubes at all comparable with that exercised by the powerful suction action of inspiration. It was noted many years ago by Dr. Charlton Bastian that in one-fifth of all cases of cirrhosis of the lung the tubes are not at all dilated. This is what might naturally be expected, as respiration would probably be feeble in many, but if the fibroid change were the essential cause of bronchiectasis it would be inexplicable.

It has been suggested that the accumulated weight of the contained secretions will suffice to distend the terminal tubes when a bronchus is compressed by an aneurysm. The terminal tubules are not the ones that are especially dilated, and the tubules lie in a closed box, the chest, and are supported outside by the same pressure as that inside them. The pressure inside the chest may be slightly increased, but there is no difference between the pressures inside and outside the bronchi, which is the important factor. The essential factors which lead to dilatation of the tubes would therefore appear to be local changes in the walls, the accumulation of secretion under tension, and the effects of inspiration on a lung which is not capable of expanding satisfactorily.

7. Fibroid thickening of the lung. There are several factors which conduce to this result: inflammatory and organizing connective tissue changes result (*a*) from the retention of the secretions, (*b*) from the prolonged collapse of the alveoli, (*c*) from the broncho-pneumonia, (*d*) from the peribronchial changes which follow on the destruction of the bronchial epithelium, which generally takes places, and finally (*e*) from the interference with the pulmonary and lymphatic circulations which normally depend on the opening up and the contraction of the alveoli. Moreover, when we bear in mind that in health the alveolar surface of each lung amounts to 100 square metres, each air cell being bathed in a film of blood 10 μ in thickness, we can realize what a difference it must make to the nutrition of the tissues when the alveoli fail to open and the amount of the circulating blood is enormously reduced; hence retrograde fibroid changes must gradually develop. The fibroid change is therefore also a natural sequence of obstruction, but the causes are not quite the same as those which produce the bronchiectasis.

8. Secondary infections producing a septic pneumonia breaking down into abscesses are not infrequent, and vary with the cause; in some they are essential and early, in others a late and accidental development.

9. Gangrene. This especially occurs with malignant disease and when the pulmonary artery is occluded. The communication between the bronchial and pulmonary arteries is a free one, but varies in different cases, and hence gangrene does not always occur even when the pulmonary artery is compressed. The condition may also result from a very virulent infection.

10. Putrefactive changes. Although the bronchi are often dilated, the contents are not always foul. The change is always a serious one, as it accelerates the destructive changes in the lung and introduces a severe toxic poison into the system; the organisms are usually anaërobic, and hence are but rarely cultivated.

11. As a secondary result of the bronchiectasis, especially if foul, there may be a pleurisy or an empyema.

Although stridor is a symptom that at once attracts attention, it is to be noted that it was present in but a very small proportion of the cases examined; it is marked when the trachea is involved, but otherwise was noticeable in only two or three of the cases of growth and aneurysm, yet severe dyspnoea was common.

Causes of Bronchial Obstruction.

Bronchial obstruction may arise from causes without or within the bronchus. Usually only one bronchus is affected, but in some cases the trachea is involved, as well as one or both bronchi. The external causes producing compression are:

1. Aneurysm of the aorta (35).
2. Malignant disease (56) of:
   - (*a*) Oesophagus, epithelioma (16);
   - (*b*) Bronchial glands—(1) primary, usually sarcoma (26); (2) secondary to growth at a distance (6).
3. Enlarged caseous or suppurating glands (12).
4. Gummata (2).
5. Abscess (3).
6. Dilated left auricle (1).

The internal causes are:

1. Bronchiolitis.
2. Syphilitic ulceration with scarring (5).
3. Primary malignant disease of a bronchus (usually columnar-celled carcinoma) (8).
4. Foreign bodies in the bronchus (8).

The material I propose to discuss is collected from our records at Guy's Hospital, and I desire to thank my colleagues for their kind permission to refer to cases under their care. I shall only discuss the general results and give a few illustrative cases.

Aneurysm of the Aorta.

During the period taken, out of 11,000 *post-mortem* examinations there have been 140 aneurysms of the thoracic aorta. Thirty compressed the left bronchus, and 16 of these eroded into it; 5 compressed the right bronchus and none eroded it. To contrast the results of compression produced by the various causes it may be noted that in this series, in 16 cases—that is, one-half—there was bronchiectasis beyond the obstruction, but in only 8 was it associated with a marked fibroid thickening of the connective tissue and increased pigmentation of the lung with a thickened pleura. In several (6) there was also an acute terminal pneumonia, and in 4 an acute septic pneumonia; in 3 there was an acute gangrene, and sometimes there was an acute pleurisy, but only once was there a pleuritic effusion. When there was no definite dilatation of the tubes the lungs generally showed bronchi full of muco-purulent secretion, often with collapse and a more or less airless lung.

The dilatation of the tubes takes place only in the part of the lung beyond the obstruction, and is irregularly distributed, being generally least marked in the anterior part. The amount varies greatly; in some cases the distension of the tubes is but slight, when the lung may be also collapsed or pneumonic. Marked increase in the fibrous tissue, as it occurs less frequently, appears to be to some extent a secondary change, and to be partly set up by the absorption of the irritating contents. In advanced cases the fibrous trabeculae stand out, the lung is very tough, and may be riddled with intercommunicating cavities. In only one in twelve was an acute gangrenous condition noted, although in many the contents were very foul. The bronchiectasis requires at least two or three months in which to develop, but occasionally it took a year or more, and it is very remarkable how very latent the constitutional symptoms may be so long as the changes are limited to the fibroid lung beyond the obstruction. When much foul secretion is expectorated, the healthy part of the lung is apt to become infected, and if an acute septic pneumonia develop, the disturbance to health is intense.

The *ascending arch* is not in very close relationship with the right bronchus, and only occasionally will an aneurysm of this part (usually a saccular one) compress the bronchus, and then on its anterior surface; not infrequently it will also involve the trachea; in none of the cases did it rupture into the bronchus. Occasionally the azygos vein, which is in close relation to the right bronchus, may also be compressed, and this may be the explanation of the only case with pleuritic effusion. In none of the right-sided cases did the breath sounds entirely disappear, nor did a local bronchiectasis develop.

A sac connected with the latter part of the *transverse arch* may compress more or less completely the left bronchus on its outer and anterior surface, and often the trachea as well, and very rarely the left pulmonary artery which lies above the bronchus (Fig. 1). This arterial stenosis probably accelerates retrograde changes in the lung and the development of the fibroid thickening, but it did not in any case lead to gangrene. In no case did an infarct form, and in only one did an aneurysm rupture into the pulmonary artery. When the aneurysm comes off lower down it will compress the bronchus from behind and on its outer side (Fig. 2).

Sometimes, however, before the bronchus is completely occluded, the lung will become over-distended, there will be a hyper-resonant note, and the distended lung may displace the heart and push down the diaphragm. That this condition may result has been almost entirely overlooked by authors, and no reference is made to it in the textbooks. Yet over-distension, we know, not infrequently results if the respiration is vigorous, when there is laryngeal obstruction in diphtheria, in tracheal obstruction from an enlarged thyroid, thymus, or aneurysm, in asthma with spasm and therefore, with stenosis of the smaller bronchi, and in acute bronchitis and bronchiolitis.

It is also what we might expect. With inspiration the chest expands and the surface of the lung closely attached to it by capillary action follows and the air is sucked past the obstruction as the lung attempts to open. With expiration there is a contraction of the lung, due to its elasticity, which is feeble, but sufficient for ordinary purposes. This drives the air from the smaller to the larger bronchi, because the pressure in the former is the greater. When forcible expiration is applied, the bronchi as well as the lung tissue are compressed with an equal pressure; if there is no obstruction the air passes on, and the pressure therefore falls in the larger bronchi. When an obstruction exists, however, this mechanism fails, the air has greater difficulty in escaping than it had in entering, and the lung becomes over-distended. The pressure may be so much raised that the diaphragm may be displaced two inches and the heart pushed out of position.

This condition gradually develops and persists for a certain time, but by degrees the bronchi fill with secretion, thus increasing the difficulties of the ingress of air, and as the contained air is not replaced, and is subjected to high pressure, it becomes absorbed by the pulmonary blood, while the lung becomes solid, airless, and dull on percussion. Lichtheim has shown experimentally that when the bronchus is completely occluded this airless condition develops almost at once.

In six of the cases the symptoms were so suggestive of pneumothorax that this diagnosis was more than once suggested.

This possibility of over-distension as a result of bronchial obstruction has been so far overlooked by all authors that the only definite recognition of the condition I have noted, is by the late Dr. Pearson Irvine, who, in the *Pathological Transactions* for 1878, recorded a case of aneurysm pressing on a bronchus in which the lung was larger than normal, although fibroid and bronchiectasic; he came to the conclusion that the result of the pressure had been first to over-distend the lung, that then the secretion had accumulated in the bronchi, and the air had been absorbed, while fibrosis and bronchiectasis from the greatly increased tension had compressed the blood vessels and impaired the nutrition of the tissues. C. Jacobsohn has also more than once noted that the position of the dullness set up by a growth at the root of the lung may vary with respiration owing to the over-distension of the affected lung, which is best shown by the *x* rays.

Case 1.—*Saccular Thoracic Aneurysm Compressing the Left Bronchus and Producing an Over-distended Lung.*

By some it was thought that the case was one of pneumothorax. The patient was a man, aged 29 years, admitted in November, 1905. He had been in the army and had had syphilis. Cough had been present for a year, and had been worse for the last three months, with dyspnoea and pain in the left chest. He had no great distress while in bed; the left chest moved badly; there was no tactile vocal fremitus, and no voice nor breath sounds on the left side. On percussion the note was hyper-resonant. There was no cardiac dullness on the left, and on the right there was dullness, which varied with respiration, extending from the fourth rib out to beyond the right nipple and down to the liver. There was marked epigastric pulsation, the heart sounds were faint, and no bruit was audible. There was compensatory breathing over the right chest. There was considerable uncertainty with regard to the diagnosis, but it was thought that most probably the condition was due to an aneurysm pressing on the left bronchus. The patient was examined with the *x* rays. The whole of the left side of the chest was remarkably transparent compared with the right; no foreign body was visible; the heart was displaced nearly 2 in. to the right of the sternum with deep inspiration, and back again behind the sternum with expiration. The diaphragm was depressed on the left side, but no trace of any aneurysm could be seen (Fig. 3). The temperature was normal and the pulse 72. There were no stridor, no evidence of any syphilitic ulceration of the trachea and bronchi, and no expectoration. It was elicited that three months previously a crust of bread had gone "the wrong way," but had this passed into the bronchus there would have been evidence before now of septic pneumonia. The extreme translucency and distension of the left chest, with the absence of any change in the aorta, threw a doubt on the diagnosis of aneurysm, and it was thought that a possible explanation of such marked translucency might be a pneumothorax, although no bruit-d'airain could be elicited, and there was no evidence on the screen of a lung compressed by air. The left axilla was explored with a Southey's tube, but no air escaped. The result of this exploration was to produce an acute attack of pleurisy, and he felt very ill for some time. His temperature two days afterwards was 99° F., and on the next day 101.6°, with a pulse of 112 and intense pain. The temperature did not reach the normal again for nine days.

In December the patient was tested with tuberculin with a negative result. Later his opsonic index to tubercle was 0.38. As the condition still remained obscure, the chest was again explored in December, with a negative result, except that there was again a sharp attack of pleurisy with pyrexia which lasted or ten days, when the condition again quieted down At

the end of 1906 there was an impairment of resonance developing at the left base; there were still an absence of air-entry, diminished tactile vocal fremitus and voice sounds. He now developed pain with sweating at night, cough, and expectoration, and at the apex behind there were distant breath sounds. A somewhat tympanitic note was noticed near the cardiac area. No tubercle bacilli could be found. During the next month or two he had a large amount of muco-purulent expectoration which was not foul. In February he began to develop slight clubbing of the fingers, and a ruh was audible over the left chest. On February 27th he had a rigor with a temperature of 105° and a leucocytosis of 33,700. He bad a sharp attack of pleurisy lasting for ten days. In March the impulse of the heart had returned to the left si3e of the sternum. There were consonating râles at the back of the left lung and not much difference between the note on the two sides.

It was at first difficult to explain the attacks of severe pleurisy which followed both attempts at exploration. It was clear afterwards that infective fluid from the distended bronchi had inoculated the pleura, as, doubtless owing to the great pressure under which it was retained, some leaked into the pleura from the puncture. Had the lung not been under tension it would probably not have set up any infection. The obstruction to the bronchus was not complete, as there was expectoration, although the movement was so feeble that we could not hear air being sucked up to the surface of the lung. It is noticeable that so long as the lung remained quiet, and before it became fibroid, so that the contents did not pass into the lymphatics but remained in the lung, or were expectorated, the patient remained free from fever, yet the faintest trace in the pleura set up a violent reaction.

His symptoms gradually quieted down, and he continued fairly well. I did not see him from April, 1906, to the end of February, 1907, when he was seized with general pains, pyrexia, and troublesome cough. Soon the expectoration became abundant and extremely foul, so that he vomited frequently. On readmission he was very ill and feeble. The left chest was contracted; the movement was deficient, there was an impaired note at the apex and below, and it was also dull in the axilla. Over the upper spaces the entry was deficient and broncho-vesicular; below it was lost. Numerous râles were audible, with compensatory breathing on the right side. The pulse was irregular, 108; the impulse was dragged ¾ in. outside the nipple by contraction of the lung. A diagnosis of gangrene of the lung was made. The condition proved fatal on the 26th, three days after admission.

Post mortem a saccular aneurysm opening just at the commencement of the descending aorta had compressed the left bronchus and produced a condition of fibrosis of the whole of the left lung with recent septic bronchopneumonia (Fig. 4). There was bronchiectasis of the tubes beyond the compression, with cavitation due to recent putrefactive infection, which was most marked in the posterior part of the lower lobe (Fig. 5).

Until I saw this case I was quite unaware that a partial compression of a bronchus was capable of producing an over-distended lung, which might be confused with pneumothorax.

CASE II.—The over-distension of a lung when there is pressure on the trachea, together with urgent dyspnoea, is well shown in Fig. 6. The patient was a man who was admitted with bronchitis and intense dyspnoea which soon proved fatal. The trachea was found to be compressed by an aneurysm of the transverse aorta.

The lung was enormously over-distended and had ruptured, producing interlobular emphysema, as shown by the lines of the interlobular septa, which, when fresh, were distended with air, but now are collapsed and empty.

I am indebted to Dr. E. W. Martin for the two following skiagrams, which excellently illustrate my contention. Fig. 7 represents a chest taken from the back. The left side is uniformly transparent; the intercostal spaces are wider than on the right. The diaphragm is flattened and displaced downwards and the heart to the right. The aneurysmal dilatation of the arch, with the marked excursion of pulsation, is well shown. There were well-marked signs of an aneurysm compressing the left bronchus. The condition was diagnosed on account of the skiagram as pneumothorax with aneurysm, but the appearance is really due to an over-distended lung. This will be at once apparent if we contrast it with an equally good skiagram of a pneumothorax (Fig. 8), also taken so long ago as 1902 by him. Here the air space is more transparent than was the over-distended lung, but the lung, shrunk up near its root, is obvious; this was wanting in the other picture. The heart and diaphragm are more displaced, and the latter has a much sharper outline.

[Since the lecture Dr. Martin has been able to find me the following notes of this case, which was in Victoria Park Hospital in 1902:

There was pulsation over the upper part of the sternum with a dull note. The note over the left chest was resonant, no cardiac dullness, the apex beat was 1 in. to the left of the sternum in the fifth space, the heart sounds were very feeble, but best heard on the right of the sternum; extremely feeble breath sounds all over the left chest—it hardly moves at all—and the whole chest, both back and front, is larger than the right. An exploring syringe was introduced in the seventh space posteriorly on the left side, and connected with a tube under water, when about thirty bubbles of air escaped.

The condition at that time was considered to be one of pneumothorax with aneurysm. The escape of the bubbles of air naturally suggested this; the skiagram, however, is definitely that of an over-distended lung, and I am supported in this view by Dr. Hugh Walsham. The air must, therefore, have escaped from the interior of the lung itself, and the explanation is that it must have been retained under great pressure, which is also indicated by the condition of the lungs.]

CASE III.—*Aneurysm Compressing the Left Bronchus: Signs at one time suggesting Pneumothorax.*

The patient was a man, aged 39 years. In October, 1906, there was pain bebind the sternum. The physical signs on admission left it doubtful whether he had pneumothorax or an obstruction of the left bronchus. There were cough and stridor, there was deficient movement of the left side, hut it looked enlarged. Some hyper-resonance was present. There were no tactile vocal fremitus, and no breath sounds, and voice sounds were diminished; on the right side there was compensatory breathing. The heart sounds were not well heard on the left side, hut were hetter than normal on the right side. On December 15th a skiagram showed slightly increased opacity in the region of the aorta. On January 7th there was no dullness; no breath sounds were audible over the left chest. In February breathing was audible behind in the upper part, but the cardiac impulse was not palpable. In March there was a hetter entry of air into the left lung, with a few râles. The impulse was in the sixth space one inch outside the nipple. On March 14th the left base was becoming dull; when the patient went out in May the left chest had an impaired note with deficient breath sounds. Fatal haemoptysis occurred a few weeks later.

CASE IV.—*Saccular Aneurysm Compressing and Eroding the Left Bronchus: High-pitched Note over the Left Chest: Signs suggestive of Pneumothorax: Fibrosis with Bronchiectasis.*

The patient, a man aged 38 years, had had cough and pain in the chest for six months. The left chest was *less flattened* than the right, there was defective movement, and a *high-pitched note* all over on the left side. There was no cardiac dullness and no dullness in the axilla. Voice sounds were deficient and breath sounds absent. Behind, the *left chest*

DESCRIPTION OF SPECIAL PLATES.

Fig. 1.—Trachea and bronchi opened from behind to show that an aneurysm of the transverse aorta presses on the anterior and outer side of the left bronchus. In this case the aneurysm is just beyond the transverse part, and has ruptured into the bronchus at A.

Fig. 2.—A similar specimen with an aneurysm of the descending aorta laid open; this is seen to lie on the posterior surface of the left bronchus, which it compresses.

Fig. 3.—Skiagram of a chest taken from the front, showing the greatly over-distended left lung, due to pressure on the bronchus from an aneurysm. The left lung is over-transparent, the diaphragm is pushed down, and the heart is pushed over to the right.

Fig 4.—The bronchi of the same case laid open from behind. A small saccular aneurysm can be seen opening from the descending aorta, compressing, and almost occluding, the left bronchus, through which a glass rod (A) has been passed.

Fig. 5.—A section of the corresponding lung, showing the resulting reduced size from fibrosis, with a general bronchiectasis. The fibrous trabeculae stand out, and there is, in addition, an acute destructive cavitation, chiefly in the posterior part of the lower lobe.

Fig. 6.—A piece of lung showing interstitial emphysema. The lines of the interlobular septa were, when fresh, distended with air, due to rupture from over-distension of the lung, produced by compression of the trachea by an aneurysm, and accompanied by intense dyspnoea.

Fig. 7.—Skiagram taken from the back, showing an over-distended left lung, due to compression of the bronchus by an aneurysm. The left chest is abnormally transparent, the aneurysm with its movement above is obvious, the heart is displaced to the right, and the diaphragm is 2 in. lower than the other side.

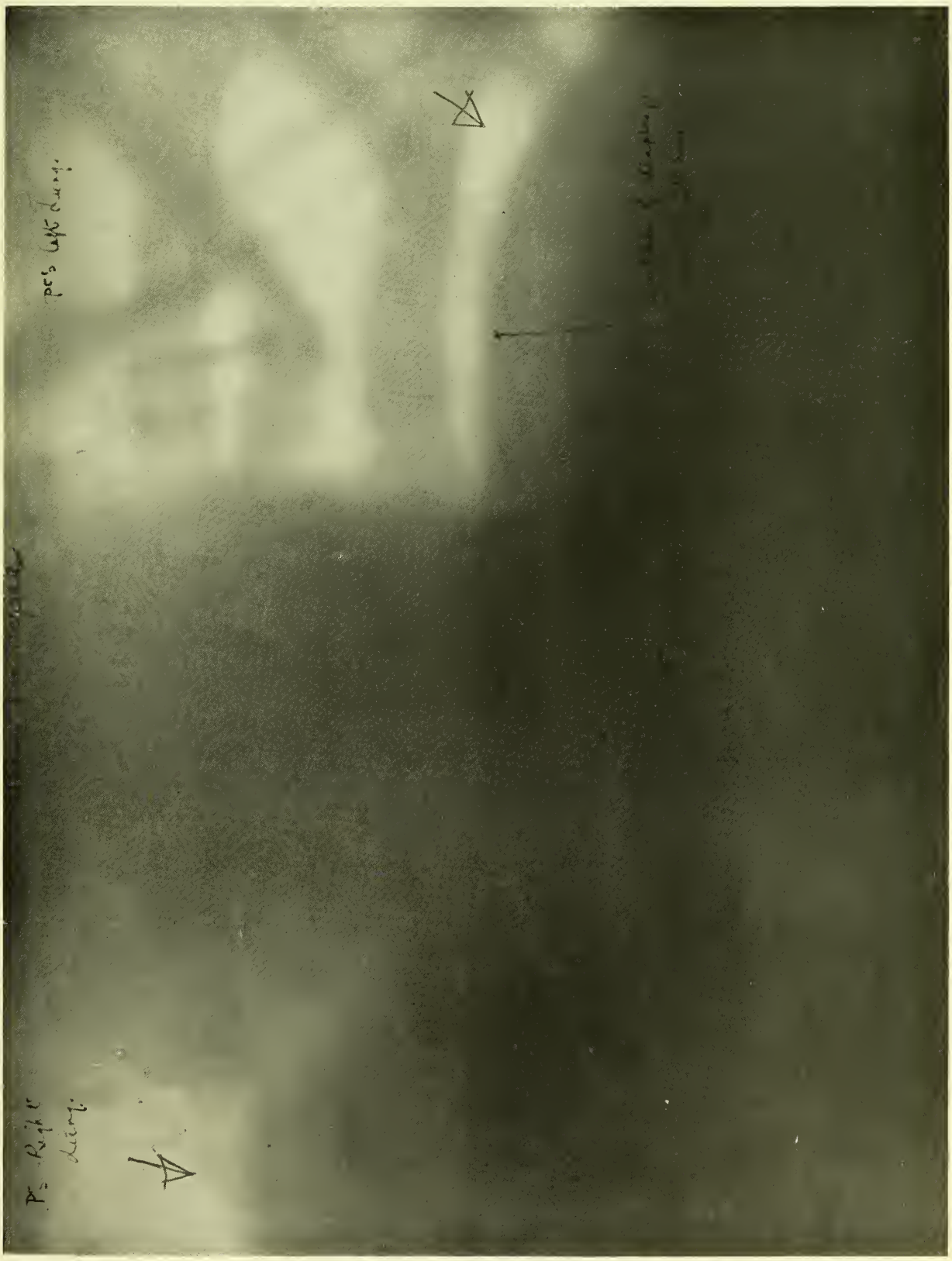


Fig. 3.—To show overdistension of left lung.

Fig. 8.—Pneumothorax for comparison.

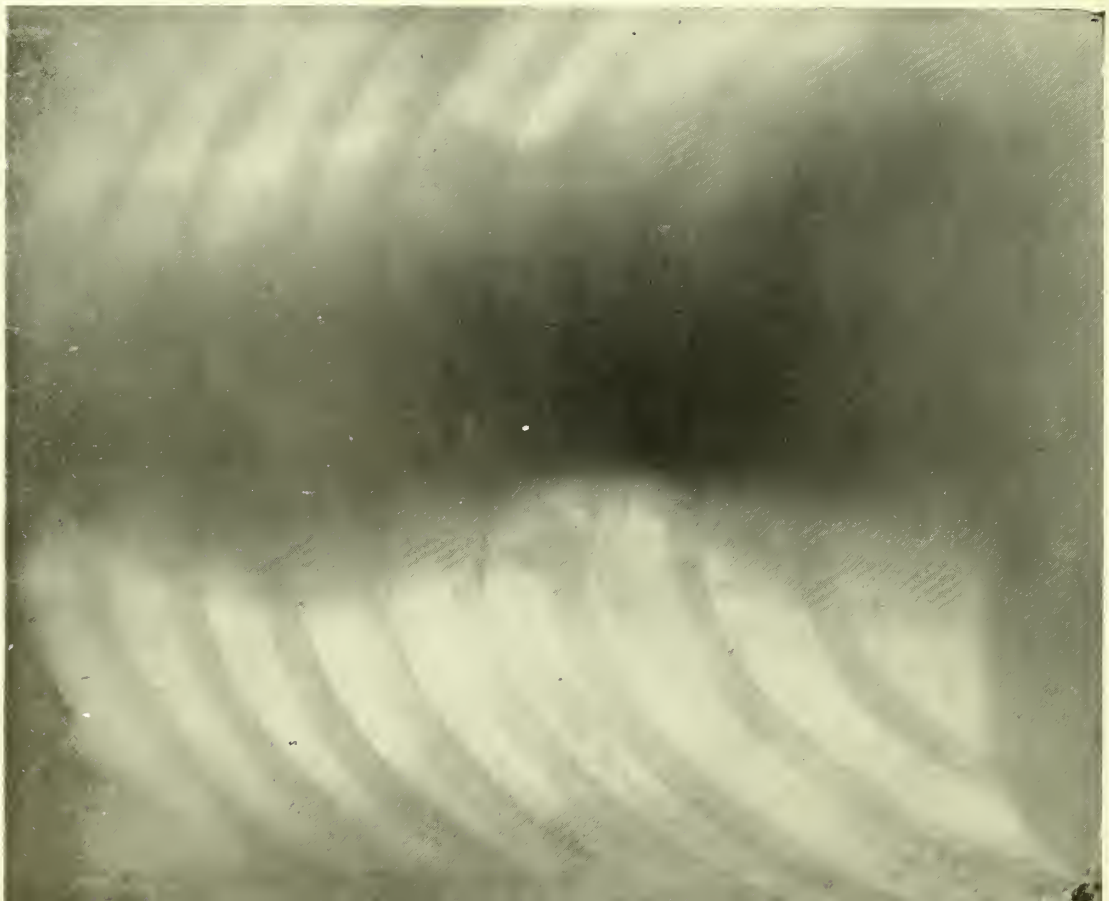


Fig. 7.—Overdistended left lung.

DR. NEWTON PITT ON THE RESULTS OF BRONCHIAL OBSTRUCTION.

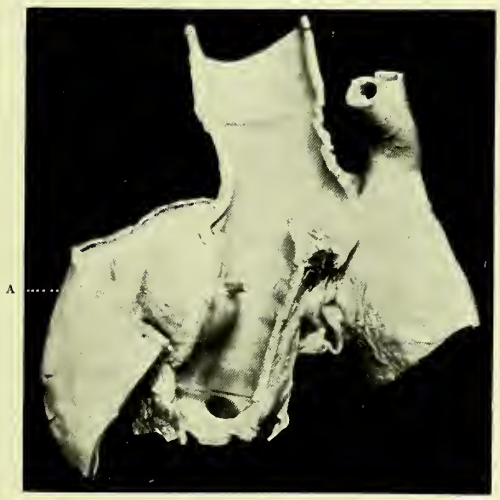


Fig. 1.

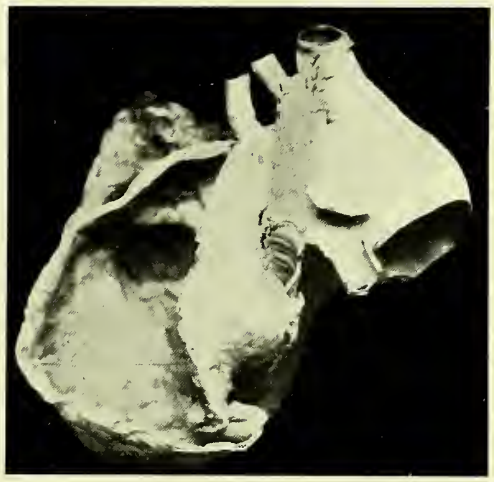


Fig. 2.

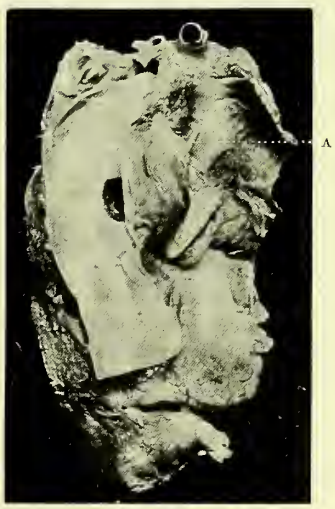


Fig. 4.

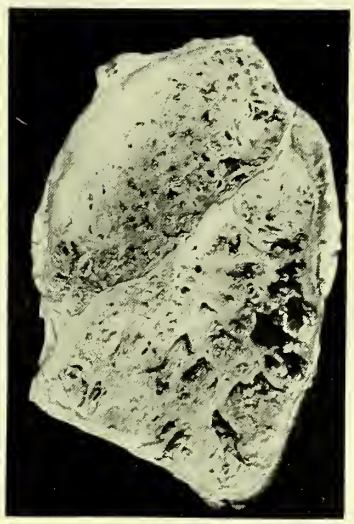


Fig. 5.

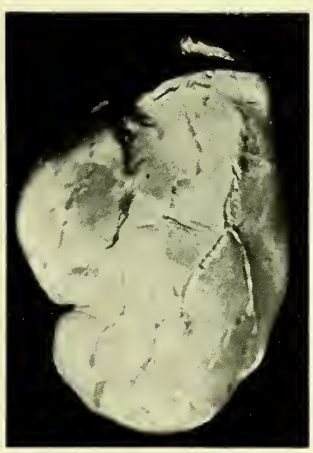


Fig. 6.

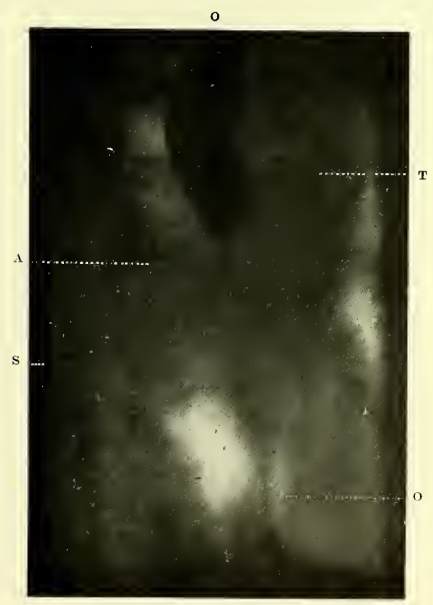


Fig. 9.

For description of figures see pp. 1848 and 1849.

DR. NEWTON PITT ON THE RESULTS OF BRONCHIAL OBSTRUCTION.

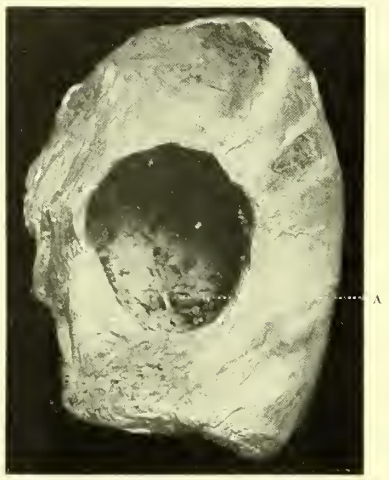


Fig. 10.

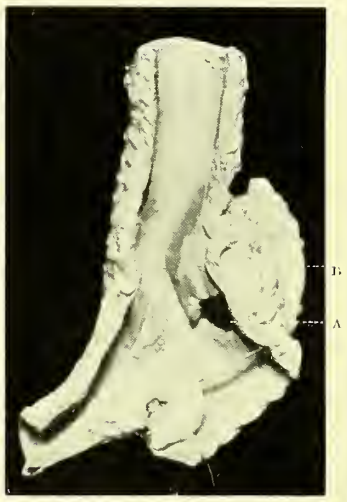


Fig. 12.

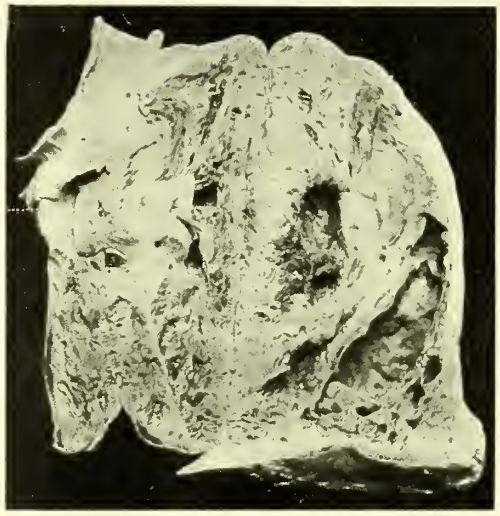


Fig. 11.

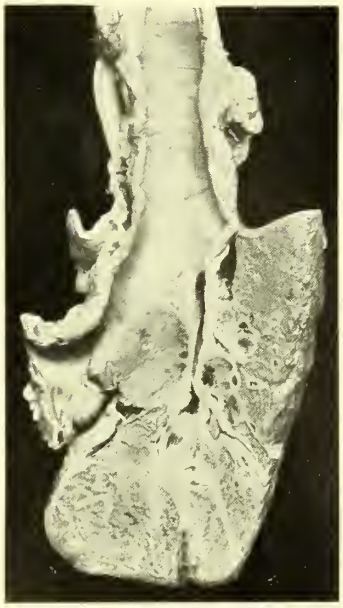


Fig. 13.

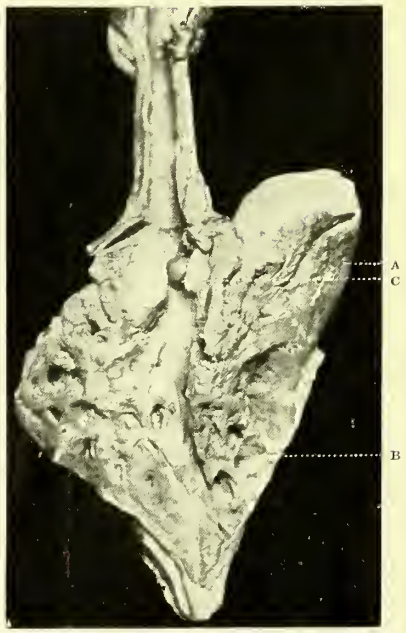


Fig. 14.

For description of figures see pp. 1848 and 1849.

bulged more than on the right; there were diminished tactile vocal fremitns and voice sounds. The chest circnmference was 36 in. and the expansion ¾ in. The heart sounds were indistinct, but were best heard behind the sternum and to the right. Diagnosis: Growth or pneumothorax. The temperature oscillated between 102° F. and normal while the patient was in the hospital. On April 26th, 1902, the chest was *smaller*, being only 34 in., and there was a slightly impaired note over the precordium. Fatal haemoptysis occurred.

Post mortem a saccular anenrysm was found opening into and compressing the left bronchus. There was a septic pneumonia of the left lower lobe, with fibrosis and bronchiectasis.

CASE V.—*Aneurysm of the Descending Aorta Opening into the Left Bronchus. ? Pneumothorax or Aneurysm.*

The patient, a man aged 38 years, had dyspnoea and pain with impaired movement and absence of breath sonnds on the left side; there was a hyper-resonant note and the heart was displaced to the right. On September 20th, 1897, there was some dullness at the left base. Fatal haemoptysis occurred while the physician was preparing to explore. ? Pneumothorax or aneurysm.

Post mortem there was an aneurysm at the commencement of the descending aorta, which had opened into the left bronchus near the point of bifurcation. The lungs were mottled with blood that had been sucked in; the edges were emphysematons. The lower lobe was solid, mottled grey-green, with a quantity of odourless pus in the tubes.

CASE VI.—*Aneurysm Compressing the Left Bronchus: Left Lung Over-distended, the Signs suggesting Pneumothorax.*

The patient in this case had a well-formed but emphysematous chest, the left side being *more bulky and larger* than the right and not moving so well; in fact, scarcely at all. No apex beat could be felt; there was a slight pulsation in the third right space 1½ in. from the edge of the sternum. There was a *hyper-resonant note* in the left chest obliterating the cardiac and the splenic dullness. Breath sounds were absent on the left side and very faint at the apex; there were no tactile vocal fremitus, no voice sounds, and no bruit d'airain. The circumference of the left chest was 18 in.; at the level of the right nipple, 17½ in.; at the level of the left, 17¾ in. There was no movement on the left. Diagnosis: Pneumothorax dne probably to the rupture of a phthisical cavity. The temperature was remittent. Later, the note at the left base was slightly impaired.

The following is a remarkable instance in which the patient survived twenty months after the first severe haemorrhage from the erosion of the aneurysm into the bronchus, the opening being probably a valvular one, which he was able to close by retracting his head and thus diminishing the antero-posterior diameter of his chest.

CASE VII.—*Aneurysm of the Aorta, one compressing the Left Bronchus, and another the Trachea: Recurrent Haemoptysis for Twenty Months before Death.*

The patient, a man aged 50, under the care of Dr. Parry Jones of Derby, had four years ago dyspnoea on exertion, when an aneurysm was suspected; twenty months before death haemoptysis (1 pint) occurred; the blood came more rapidly when he leaned forwards, and was checked when he drew back his head. He also noticed that turning his head to the right reduced the volume of his right pulse. Gradually a slight pulsation with a diastolic jog developed beneath the left clavicle. There were diminution of breath and voice sounds on the left side, bnt no dullness. There were four further attacks of haemoptysis amounting to several onnces during the next year, but by reducing his fluid to 13 oz. and keeping qniet he kept them in abeyance.

Three dilatations were found in the ascending, transverse, and descending portions of the arch. The opening into the air tube was closed up. There was no great amount of bronchiectasis. It is remarkable he should have survived the first rupture for twenty months.

CASE VIII.—*Saccular Aneurysm of the Descending Aorta which Compressed the Left Bronchus and the Oesophagus.*

The patient, a man aged 49, under the care of Dr. Morgan of Seaford, had had pain for a year in the left chest and cough for three months. Recently there had been expectoration in the morning streaked with blood. He had dysphagia, liquid food frequently coming back again into his mouth after some little time if he swallowed. Dyspnoea ensued on exertion; there was defective movement on the left side, with distension of the veins on that side of the chest. There was resonance with deficient breath sounds over the left side, and a very marked delay (almost one second) in the onset of inspiration, which was quite noticeable when a double stetboscope was used. There were deficient voice sounds and the tactile vocal fremitns note was very slightly impaired, if at all. In the skiagram (Fig. 9) a large saccular aneurysm can be seen near the origin of the descending aorta, pressing on the left bronchus and delaying the passage of some milk and bismuth he had just taken. The oesophagus can be seen distended above and the bismuth trickling past the obstruction. When he contracted the oesophagus some of the contents were returned into his mouth.

DESCRIPTION OF SPECIAL PLATES.

Fig 8.—A skiagram taken from the back of a case of pneumothorax, due to rupture of an early phthisical cavity, for comparison. The lung, contracted up at its root, is visible above the very clear area occupied by air; the outline of the diaphragm is more distinct than in Fig 7; the heart is more displaced. The low flat condition of the left diaphragm, which is displaced down, is obvious.

Fig. 9.—A skiagram of an oblique view, showing an oesophagus, o, with its upper part distended with bismuth and milk. The spine, s, lies to the left, and the shadow of a saccular aneurysm, A, extending up to it, can be seen compressing the oesophagus; below this point a linear trickle of bismuth alone passes; the shadow extends higher than the point of compression: T is the dilated thoracic aorta.

Fig. 10.—The left lung, containing a cavity 4 in. across. In removal the anterior wall was torn and the walls when fresh were flocculent, but have been made smooth, unfortunately, in preparing the specimen. A probe, A, can be seen projecting through the remains of the bronchus; the pulmonary artery was also involved. An epitheliomatous growth invaded the root and slowly produced this gangrenous cavity, which, owing to the absence of breath sounds, gave no signs during life.

Fig. 11.—A section of a right lung which is fibroid, with bronchiectasis and gangrene, due to infiltration of the root by a sarcoma which compressed and almost obliterated the pulmonary artery and the bronchus; a probe, A, indicates the lumen of the bronchus. The lower lobe is greatly reduced in size and excavated by a gangrenous cavity.

Fig. 12.—A trachea and bronchi laid open from behind, showing the opening, A, through which a suppurating gland, B, had discharged into the right bronchus.

Fig. 13.—A trachea, with part of the right lung. The trachea and bronchi are scarred with syphilitic ulceration, the right bronchus is markedly narrowed, the lung is dense and fibroid, but there is no bronchiectasis.

Fig. 14.—A trachea and right lung, showing a pea, C, tightly wedged in the lower branch of the right bronchus. The upper lobe, A, is healthy, but the lower, B, is riddled with smooth-walled intercommunicating cavities. There is marked bronchiectasis, with fibroid replacement of the lung tissue in the lower lobe.

The features especially associated with the pressure of an aneurysm on a bronchus may be summed up as follows: 1. The left bronchus is involved six times as often as the right. 2 The right is very rarely eroded. 3. Resonance with silence. There are deficient or absent breath sounds over the left lung with a resonant or hyper-resonant lung. This does not necessarily indicate more than a partial occlusion of the bronchus, and occasionally in an early stage the left lung may be over-distended and be too transparent to *x* rays. The condition has, then, to be diagnosed from pneumothorax. 4. With *x* rays dilatation of the aortic arch can generally be detected, as the disease of the wall is generally multiple and diffuse. The local aneurysm often is best shown in a view in the half turned position with the patient sitting up. The deficient and delayed movement of the lung, the alteration in its density, and the abnormal position and movement of the diaphragm are usually obvious. Occasionally no local aneurysm nor yielding of the aorta can be detected. 5. Later, the development of an impaired note rather than complete dullness, with gradual development of bronchiectasis. It is remarkable how the amount of change which develops is always under-estimated owing to the breath sounds being absent or feeble. 6. Absence of pleuritic effusion. 7. Retardation of the onset of inspiration on the affected side. 8. In nearly half the cases more or less haemoptysis. Pain with dyspnoea on exertion are very constant symptoms.

As soon as the mucous membrane ulcerates the liability of the contents of the bronchi to become freshly infected is greatly increased, as the spot is very apt to become a nidus for organisms itself. Hence necrotic changes in the lung occur more often when the tube is ulcerated than when it is only compressed, just as primary growths in the bronchus are especially associated with gangrene.

MALIGNANT DISEASE (55 CASES).

The growth may originate (1) in the bronchus, (2) in the glands at the root of the lung, (3) it may be a secondary

deposit in the glands at the root of the lung and grow into it along the bronchus, and (4) as an epithelioma of the oesophagus ultimately invading the bronchus.

1. Primary growth in a bronchus is rare (8); in 4 cases it started in the right and in an equal number in the left. The growth is generally a columnar-celled carcinoma—at least four-fifths of cases of primary cancer of the lung start in the bronchial mucous membrane. Most of the cases came under observation for pleuritic effusion, which not infrequently required tapping more than once. In half the cases gangrene of the lung developed. In some a fragment of growth was expectorated, and thereby its malignant nature determined. There are generally pain and pressure signs.

2. Growth in the glands (26 cases) half affecting each bronchus. Two were described as lymphadenoma, but as they eroded the bronchus it is more probable that they also were sarcomatous.

3. Secondary deposits in the glands at the root of the lung (6 cases): the growth compressed the bronchus and invaded the lung.

4. Epithelioma of the oesophagus (16 cases). The two bronchi are invaded with equal frequency. In four they were only compressed; in the others the growth had fungated in. With epithelioma the pulmonary condition rapidly proves fatal, with a septic pneumonia, abscess formation, or gangrene. Bronchiectasis and fibrosis have not time to develop. The rapidly fatal changes are due to the infection with food from the communication with the oesophagus. The food is doubtless often squeezed in by the contraction of the oesophagus owing to the obstruction of its tube beyond by growth. In one case a deposit in a gland led to compression of the lower branch of the left bronchus with the development of bronchiectasis beyond. A troublesome paroxysmal cough, pyrexia, and pain are the indications of the onset of trouble in the lung, to which foul expectoration is soon added.

The special features of malignant disease are the frequency with which pleuritic effusion and also gangrene develop (more than half). The effusion is an early sign, and both this and the collapse of the lung, as well as the growth itself, all give dullness which is often quite marked. It has been pointed out by writers that the physical signs given by the changes in the lung and the pleura are obvious at an early stage and tend to obscure the evidence of the growth itself. In many the pulmonary artery is also compressed and may be occluded, and this is much more frequently so than by an aneurysm; hence the rapid development of extensive patches of gangrene which may lead to the development of large cavities, often without fibroid thickening of the wall. In a small proportion fibroid changes in the lung are well marked; in others there has not been time for them to take place. Bronchiectasis is by no means always present, but only in one third.

The massive infiltration of the root of the lung occludes not infrequently the lymphatics and blood vessels as well as the bronchi; hence oedema of the pulmonary tissue tends to develop and the bronchi to become more rapidly overfull of secretion. Stridor is not infrequently present, especially on exertion, but is most marked when the growth has also compressed the trachea.

CASE IX.—*Squamous Epithelioma of the Root of the Lung with Destructive Disease of the Lung beyond.* (Fig. 10.)

The patient was a man, aged 65 years, who for twenty months expectorated material of the colour of strawberries and cream, at one time amounting to a pint a day. The only physical signs were deficient breath sounds over the left base.

Post mortem the left lower lobe of the lung was excavated out into a cavity 4½ in. across, with flocculent necrotic walls, there were numerous dilated bronchi in the rest of the lobe with fibrosis. The bronchus and pulmonary artery were compressed, being embedded in the mass of growth. The primary seat was not determined as the *post-mortem* examination was limited to the thorax.

This specimen is of interest as showing the early formation of a single large cavity from destructive changes from necrosis, and in this instance without any foul secondary putrefactive changes. It illustrates the fact that the physical signs of a large cavity over 4 in. across may be almost *nil* when there is stenosis of the bronchus, owing to the absence of any breath sounds. Contrast it with the extensive fibrosis and the usual absence of large cavities which result when the pressure is due to an aneurysm.

As another example showing how a mass of sarcoma growing into the root of the lung associated with occlusion of the pulmonary artery tends to form large cavities, in addition to a general fibrosis of the lung with moderate dilatation of the tubes, the following may be quoted:

CASE X.

The patient, a woman aged 38 years, died a few hours after admission. At the inspection the left lung was healthy. A section has been made of the right lung, with the mass of growth at its root. A rod has been passed through the much-constricted bronchus. The pulmonary artery except for a small branch was occluded. Almost the whole of the lower lobe was excavated out into a cavity. There were smaller cavities in the lower part of the upper and middle lobes with general dilatation of the tubes in a dense fibroid tissue. (Fig. 11.)

CASE XI.—*Primary Cancer of Bronchus: Expectoration of a Nodule of Growth: Duration of Symptoms three and a half Years; Fibrosis with Bronchiectasis.*

The patient, a man aged 40 years, first noticed some wheeziness in September, 1901, and during the year 1902 he became short of breath on exertion; he lost half a stone in weight; there was an ounce of muco-purulent secretion daily, and on exertion or deep breathing there was some stridor. The wheeze or musical whistle with expiration was loudest when he lay on his left side. He had no pain. In October there were deficient breath sounds with a slight squeak, and deficient voice and tactile vocal fremitus over the left side, with dullness in front at the apex. There was similar deficiency in breath sounds at the apex; the base was fairly normal; the right side was quite normal. With a skiagram there was a slight opacity at the left apex, but less than is often met with in growth, although the evidence pointed in that direction. There was no evidence of aneurysm. He was subject to attacks of pyrexia with an increase in the expectoration. He continued to lose weight, and towards the end of the year there was a pleuritic rub. He had a few slight rigors. In December the dyspnoea had increased considerably, and he was very frequently sick unless very careful with his diet. A rough, blowing systolic bruit developed to the left and above the heart; the resonance became more impaired above, and the breathing indeterminate, with an increase in the voice sounds. The impulse of the heart was pulled upwards and outwards. There was pulsation over the affected area at the left apex which apparently was transmitted from the heart and was not due to an aneurysm. The diaphragm was as high as the fifth rib in the axilla. During the year 1903 he was able to get about if he moved slowly. Expectoration was limited to half an ounce; it was slightly blood-stained, but never offensive. The amount of blood varied somewhat.

The patient continued fairly well until May, 1904, when he had an attack of pericarditis, which lasted for six weeks. In December, 1904, he coughed up a piece of tissue an inch long and three-eighths of an inch in diameter, which came away quite easily with only a little haemorrhage. On microscopical examination this was found to be a portion of glandular carcinoma composed of tortuous columns of columnar cells; there were also cells in alveoli which communicated with these columns. Another nodule was expectorated in the following March. On April 18th, 1905, I again saw him with Dr. Cockburn of Ealing. The patient had got worse during the last six months, frequently expectorating as much as an ounce or two of blood. The dyspnoea had increased. He was practically confined to his bed. For a long time he was unable to lie on his left side, and latterly only on his right side. There was pulsation as high as the third space above the nipple, to the left of the heart; there were a noisy, wheezy stridor and noisy expectoration. We came to the conclusion that a nodule of the growth had fungated through, probably into the trachea, and it was a question whether there was not originally a foreign body in the left bronchus, which was the starting point of his trouble. He died the next day.

On *post-mortem* examination the right lung was found over-distended, but otherwise normal; there was an adherent pericardium. There was a mass of growth at the root of the left lung, in which the bronchus and vessels were imbedded. No foreign body was found, the left lung was extremely contracted. The upper lobe was reduced to a mass about 2 in. across and 7 in. long, riddled with small bronchiectasic cavities, while the lower lobe was converted into a very dense fibroid mass no bigger than the lung of a child of 6, and was also riddled with cavities.

COMPRESSION BY INFLAMED GLANDS (12 CASES).

The cases were: Enlarged glands 7, suppurating and opening into bronchus 5. These cases occur mainly in infants in whom the glands are active, and the antero-posterior diameter of the thorax is so small that a mass of glands at the bifurcation of the trachea may lead to serious symptoms, but especially when they press on the trachea. They less often compress the bronchi seriously, and this generally only occurs when there are enlarged glands on each side of the bronchus, which accounts for the comparatively few cases in which enlarged caseous glands give rise to pressure symptoms. The cases of pressure on the trachea are apt to be looked upon as

due to laryngeal diphtheria, but the absence of movement of the larynx is of diagnostic importance. Their enlargement may be made out by dullness internal to one scapula, by the opacity shown by *x* rays, by the absence of breath sounds on one side, and by a venous hum over the sternum on retracting the head, although I have only been able to elicit this sign three or four times.

When a suppurating gland breaks into a bronchus the child has severe dyspnoea with violent paroxysms of cough, and may bring up some purulent secretion, while there is soon an active development in the lung beyond of fresh physical signs. When in a child there are evidences that a bronchus is compressed, it is far more likely to be due to an enlarged caseous gland than to a foreign body, unless there is a definite history of sudden onset of symptoms after swallowing something Most of the cases due to caseous glands are complicated with phthisis; less than half of them are diagnosed, but *x* rays make it much easier to do so now than formerly.

In 3 cases abscesses opened into a bronchus: (1) empyema, right side; (2) suppurating bronchial glands secondary to suppurative pylephlebitis; and (3) abscess communicating with the oesophagus and gangrenous cavity in the lung.

Case xii.—*Compression of Trachea and Right Bronchus by Enlarged Caseous Gland.*

A child, aged 2 years, was admitted to hospital in 1910 with dyspnoea and stridor, which was supposed to be due to laryngeal diphtheria. On examination it was found that there were deficient breath sounds over the right side, with impaired resonance. The larynx did not move with respiration, and compression of the root of the right lung by caseous glands was diagnosed. The child died a few hours later. A large caseous gland compressed the trachea in front, just above the bifurcation. Another large gland between the bronchi was softening; the right lung was collapsed and airless; the left lung was normal. (Fig. 12.)

Dilated Left Auricle.

A cause of pressure sufficient sometimes to cause physical signs during life, but rarely to present much evidence *post mortem*, is a dilated left auricle due to mitral stenosis, to which attention was directed many years ago by Dr. Wilkinson King.

Case xiii.—*Pressure on Left Bronchus by Dilated Left Auricle.*

In 1888 a woman, aged 50 years, was seen suffering from mitral stenosis with a greatly dilated left auricle. At the inspection there was also a softening *ante-mortem* clot in the appendix. The left base was airless and sank in water; it could be inflated less than the right one, which was also oedematous and engorged. Both were deficient in air. The dilated left auricle markedly compressed the left bronchus.

Fibrous Bronchiolitis due to Irritant Gases.

Inhalation of irritant gases is a well-known cause of intense dyspnoea and bronchitis, not infrequently fatal, with a greatly over-distended lung, of which we have had examples from time to time. Fraenkel has shown that this condition may be due to an acute fibrous obliterating bronchiolitis, which may develop within a fortnight. He has recorded cases after inhalation of the fumes of chlorine, of cow hair and lime dust, and after calcium chloride dust. A man after inhaling chlorine had intense dyspnoea, which passed off but returned next day, when he was cyanosed, with an extremely distended lung and fine crepitations. The condition again cleared up, to return again after a week, when he died. Widespread fibrous peribronchial inflammation was found round the smaller bronchioles, with necrosis of the lining epithelium, producing constriction of the tubes with dilatation beyond. Schmorles has described a similar condition in the bronchopneumonia of measles, with obliteration of the bronchioles. The ingravescent cyanosis with dyspnoea and a rapid and great increase in the size of the lung are the essential points, especially when there has been exposure to irritant gases.

Stenosis of the Bronchi from Syphilis.

Out of 7 cases, 2 were due to gummata pressing on a bronchus. The way in which the tubes dilate under violent inspiratory efforts with bronchial obstruction is well shown in a case (No. xiv) of a man, aged 64 years, where a gumma pressed on the upper division of the right bronchus. The upper and middle lobes were over-distended; the lower lobe was airless from compression with five pints of fluid. The illness was rapidly fatal. The second (No. xv) was a chronic case with a gumma compressing the superior cava and the right bronchus; the bronchi beyond were dilated up to their extremity, and their walls were thickened; the lung was solid. There was extensive pleuritic effusion into the left side secondary to the pressure on the cava.

There were 5 cases of cicatricial stenosis of the bronchus secondary to syphilitic ulceration, and the patients were all under the age of 40. In 2 the larynx was also involved, in 4 the trachea, and in 4 there was bronchiectasis. Stridor is generally present, but often is independent of the bronchial lesion. There are generally other signs of syphilis present, so that an accurate diagnosis can be made in the majority of cases.

Case xvi.

The patient was a man, aged 38 years; he was admitted with extreme laryngeal stridor and fixed cords due to laryngeal paralysis requiring tracheotomy. *Post mortem* the left lower lobe was collapsed. There was sypbilitic stenosis of the right bronchus, with scars on the trachea, fibroid right lung, with dense gummatous fibrous mass involving the root of the lung and the vagus. In spite of the vagus being involved, there was no destructive disease of the lung. Sir W. Gull at one time suggested that the destructive lung disease associated with aneurysm was due to implication of the vagus. (Fig. 13.)

Foreign Bodies.

The result of the introduction of a foreign body into a bronchus depends upon whether the bronchus is partially or completely occluded, on the vigour of respiration, and on whether the body is an organic one which will readily putrefy.

Our knowledge of the changes which result largely depend on the experiments of Lichtheim in 1878, who introduced laminaria tents into the bronchi of rabbits, and also in others removed portions of the third and fourth left ribs, ligatured the bronchus, and studied the results. He showed that when the plug completely occluded the bronchus the lung quickly collapsed and became airless. There was at once an intense dyspnoea which gradually quieted down. In many cases the animals died within twenty-four hours with pneumothorax of the opposite side from rupture of an enormously over-distended lung; in others the over-distension of the sound lung had stretched the capillaries and made them useless, equally producing a fatal result. Pneumothorax from this cause does not occur in human beings. The affected lung beyond the obstruction was of a dull brown-red colour, with the consistency of muscle. With early death the bronchi were empty, but became filled with a thick white mucoid secretion whenever the animal did not die too soon. When he ligatured the bronchus alone the same result was produced.

He found that when the animal was made to breathe oxygen absorption was much quicker, in some cases being under an hour; CO_2 absorption was less rapid, but much more so than when it breathed air. When the pulmonary artery was also ligatured absorption was much delayed, showing that the gases were absorbed by the blood circulating in the lung.

When the chest was merely opened and the lung exposed to the air atelectasis took place quickly, but did not if the pulmonary artery was ligatured. This opening of the chest vitiates many of his results, as it prevented any vigorous respiratory movements from taking place. This was well shown by the fact that when the bronchus was ligatured the opposite lung did not become over-distended to the extent it did when the bronchus was blocked by a foreign body. When the hilum of the lung, including the pulmonary artery, was ligatured there was generally no infarct, because there had been no suction of the blood owing to the absence of the opening up of the alveoli with inspiration. This affords the explanation of the absence of infarcts in cases of growth at the root of the lung and of aneurysm in which the pulmonary artery had been occluded by pressure. When the animal survived some weeks the lung was fibroid and converted into a sac of dilated tubes, presenting an appearance similar to that of a calculous pyelonephritic kidney.

Partial obstruction of the bronchi has not been thoroughly studied experimentally, and we have still much to learn

the presence of expectoration in amount shows that this is the more usual condition which results from the presence of a foreign body in a bronchus, and I have little doubt that in some cases the lung at first becomes over-distended, and is not always airless and collapsed.

Schuller has shown that it is possible to introduce clean food into the bronchi of rabbits, and if it can disintegrate it will disappear with no change in the lung beyond that of catarrh; when it cannot, fibroid changes with bronchiectasis result. When dirt, however, was mixed with the food gangrene or septic pneumonia resulted.

There have been the following cases: (1) Head of barley grass in left bronchus 129 days; (2) pork in right bronchus possibly for six months; (3) woolen top of toy in left bronchus; (4) pea in left bronchus; (5) feather after tracheotomy in right bronchus, but not cause of death; (6) pipe stem in bronchus after fatal fall down ladder (not cause of death); (7) newspaper in trachea, introduced intentionally; and (8) sponges in trachea, introduced intentionally.

The following case presents many very remarkable features. The boy swallowed a head of barley grass during a paroxysm of coughing, and this passed into his left bronchus, where it remained 129 days. He developed within two days after the entrance of the body an acute pneumonia which became gangrenous, and he had an extremely prolonged dangerous illness. Acid-fast bacilli were found on one occasion in the sputum, and were assumed to be tubercle bacilli, but he made an excellent recovery as soon as he expectorated the piece of grass. He never reacted specifically to tuberculin. He has maintained perfect health for the five years since, and is now strong and well.

It is very rare for any one to make such an excellent recovery after such extensive mischief in the lung, and it is suggested that the acid-fast bacilli came from the grass, as these grasses are said to contain them.

CASE XVII.—*Ear of Barley Grass in the Left Bronchus for 129 Days: Gangrene of the Lung: Complete Recovery.*

A boy aged 12 years, while recovering from an attack of whooping-cough, was playing with a head of grass in his mouth, when he was seized with an attack of coughing and the piece of grass disappeared. He tried in vain to make himself sick. On the next day the temperature was 102.8°, and on July 25th—the following day—Dr. J. E. G. Calverley of Folkestone, under whose care he was, found that the temperature was 105.2°, with a pulse of 100. There was pain in the epigastrium. The entry of air at the left apex in front was deficient. On the 28th there was diminished entry over the same area, with diminished movement but a *hyper-resonant* note. There was tracheal wheezing, conducted over the whole chest as rhonchi. The temperature, respiration, and pulse remained high, and on the 30th the boy had consolidation at the left apex down to the third rib, with bronchial breathing and pectoriloquy. The larynx was found to be normal. On August 1st there was profuse epistaxis, with a tympanitic note at the left apex in front, and down to the level of the fourth rib in the axilla. The entry of air remained deficient at the left apex. He became anaemic from recurrent epistaxis. He then passed under the care of Dr. R. B. Wrightson at Aldeburgh. The temperature gradually dropped to 99°, and the boy was able to get about. The sputum at first was frothy, but later became abundant and mucopurulent, and was said to contain tubercle bacilli. In the middle of September his temperature ran up to 104° and he developed signs of consolidation at the left base. In the upper lobe there was dullness at the apex with signs of a cavity in front in the second and third spaces. There was a marked oscillation of the temperature with very foul breath indicative of gangrene. He was extremely ill for a long time with a very feeble pulse. He took creosote regularly and was well nursed in the open air, so that gradually he recovered his health. No tubercle bacilli could be again detected, and at the end of October he had gained a stone in weight. The sputum was reduced to ½ oz. in amount daily, and was no longer foul. On November 30th, 129 days after swallowing the grass, he had a fit of coughing, and brought up a head of barley grass 1¼ in. long from the left bronchus. The long awns had broken off from decomposition, and so it was able to pass out again backwards, which had been quite impossible so long as they were intact. There was a gradual retrocession of the signs, but there remained at the apex a deficient resonance, with indeterminate breathing and increased voice sounds; a few moist sounds persisted at the lower part of the lung for some time. In December the opsonic index for tubercle was found to be 0.6 and 0.65 on two occasions. An attempt was made to see whether any acid-fast bacilli could be cultivated from the piece of grass, but none could be found, although many other organisms were present.

Fig. 14 (Case XVIII) shows a pea wedged in the lower bronchus on the right side. The upper part of the lung is healthy, but the rest of the lung is densely fibroid and riddled with intercommunicating cavities. The patient was a boy, aged 3 years, with cough and vomiting. He died with empyema and cerebral abscesses a month later.

CASE XIX.—*Piece of Pork in Right Bronchus: Bronchiectasis: Septic Pneumonia.*

In 1888 a man, aged 60 years, was admitted. For six months there had been wasting, and for two weeks foul breath. For two days there had been deficient movement of the lung on the right side, pleural rub in the axilla and base, deficient air entry, râles, with amphoric breathing over the middle lobe.

At the inspection the right lung was fibroid with general bronchiectasis of the main bronchi. Low down was found a piece of pork ½ × ⅜ in. There were cavities in the lower lobe. There was acute pneumonia at the lower part of the upper lobe with oedema of the left lung. Had the pork been there for six months?

CASE XX.—*Piece of Wood in Left Bronchus: Bronchiectasis of Both Lungs.*

In 1905 a female child, aged 16 months, was seen. She was well until five weeks before admittance, when she had a cough and dyspnoea. The right lung was resonant on the anterior part in front, but impaired behind. There were crackling râles, bronchial breathing, and pectoriloquy behind.

At the inspection there was advanced fibrosis with bronchiectasis of the right lower, the right middle, and the left lower portions, with a mass of dilated tubes in the midst of the parts of the lung which were fibroid. The *two left upper lobes* were distended; there was recent bronchopneumonia. In the left bronchus, just beyond the point of bifurcation, there was a smooth piece of wood broken off a toy. It was doubted at the necropsy whether the piece of wood had caused the bronchiectasis, because it was found in the left bronchus and the bronchiectasis was also on the right side, but probably at an earlier stage it was in the right bronchus, and had produced changes on that side and been displaced to the left later.

Although many cases of foreign bodies in the bronchi have been recorded, in but very few has an accurate note been made of the physical signs in the lung in an early stage of the illness. The body generally passes into the right bronchus because it is more vertical and is also slightly larger. The changes are unilateral, serious, and progressive.

The following are the most important evidences suggestive of a foreign body in a bronchus, and in any such case a bronchoscope should be passed in order that the condition of the tubes may be determined, and a positive opinion formed: (1) A most important factor in diagnosis, and one which until we had *x* rays was absolutely essential, is the history of swallowing something immediately before the onset of an acute attack of severe dyspnoea and cough. (2) Not infrequently a whistling or wheezing sound which the patient can localize to one side, varying in quality if the object is not fixed in the bronchus. (3) Great distress, with not infrequently a constricting pain behind the sternum, made worse by movement. (4) Dyspnoea, with varying exacerbations, often intense and made worse by cough, but it may be elicited only by exertion. After a time there may be no distress. (5) Absent or, rarely, very noisy breath sounds, with some slight diminution of voice and tactile vocal fremitus limited to one side of the chest. (6) At first resonance on the affected side, soon consolidation with dullness, and, after a time, the signs of cavities, and the simulation of phthisis; query occasionally hyper-resonance. The physical signs sometimes vary from time to time. (7) With *x* rays, the defective movement of the diaphragm and of the thorax, the alteration in the density of the lung, and not infrequently the outline of the foreign body itself, may be clearly made out. (8) Delay in the commencement of inspiration as compared with that on the other side. (9) Defective movement with consequent diminution in the size of the affected side. (10) Violent paroxysmal persistent cough, which is apparently most marked when the object is at the bifurcation, as the sensibility of the mucous membrane is greatest here, as can be noticed when passing a bronchoscope. (11) Early pyrexia, which later becomes hectic, associated with chills. (12) After a short time an expectoration more or less profuse, purulent, often bloody, and later often fetid. (13) In some there are nausea and vomiting, which may be very troublesome. (14) Pneumonia often develops in forty-eight hours; this may be septic and may be followed by gangrene.

[I am indebted to Messrs. Bale, Sons and Danielsson for the loan of the block of the portrait of Dr. Bradshaw which appears on p. 1.]

ABSTRACTS OF CASES OF BRONCHIAL OBSTRUCTION AT GUY'S HOSPITAL BETWEEN 1887 AND 1909.

ANEURYSMS COMPRESSING RIGHT BRONCHUS.

CASE XXI.—*Aneurysm compressing Right Bronchus: Pleuritic Effusion.*

1901. Woman, aged 45. For fourteen months she has had pain in the right shoulder and arm. Dyspnoea for eight months. There was a pulsating suprasternal tumour. Oedema of right chest; 3½ pints of fluid were drawn off from the right chest.

Post Mortem.—The right upper lobe was compressed by a saccular aneurysm of the ascending aorta, which also pressed upon the right bronchus and the trachea.

CASE XXII.—*Aneurysm compressing Right Bronchus.*

1898. Man, aged 37. There was pain with dyspnoea and dysphagia. The veins over right chest were distended.

Post Mortem.—There was a large saccular aneurysm containing about 10 oz. of clot opening into aorta from behind and 1½ in. above the valves. The right lung and right bronchus were slightly compressed. There was inflammatory oedema of the right base, and all the tubes up to the smallest on both sides were full of muco-pus.

CASE XXIII.—*Aneurysm pressing on Right Bronchus.*

1899. Man, aged 26. Was admitted moribund owing to an aneurysm having ruptured into the *left* (?) pleura. There was slight pressure on the right bronchus; the lungs were oedematous.

Post Mortem.—There was a saccular aneurysm on the right side of the aortic arch; the opening was small.

CASE XXIV.—*Aneurysm of Ascending Aorta compressing Right Bronchus.*

1905. Man, aged 36. Pain in right side five months; dilated veins; dyspnoea. There was a dull, pulsating tumour at the right of sternum with aortic incompetence. Nothing abnormal in the left lung on admission. November 11th, 1904: Deficient breath and voice sounds on the right side. Gelatine injections were administered for some weeks with benefit. Two skiagrams showed the right chest *very transparent*. In May, 1905, he had chronic bronchitis with broncho-vesicular breathing over the right lower lobe. He derived great relief from venesection. There was occasionally a little pyrexia. Death occurred from exhaustion, October 22nd, 1905.

Post Mortem.—Fusiform aneurysm of ascending and transverse arch, compressing trachea and right bronchus upper lobe right lung airless.

CASE XXV.—*Aneurysm pressing on the Right Bronchus.*

1902. Man, aged 47. For five months pain and swelling over the right chest and some pulsation to the right of the sternum, diminished vesicular murmur over the right side, and increased on the left; the note became dull at the right base, and the breath sounds finally disappeared.

Post Mortem.—The right bronchus was compressed by a saccular aneurysm of the ascending arch. There was an excess of muco-pus in the trachea, in the bronchi, and especially in the bronchioles of the right lung.

CASE XXVI.—*Aneurysm opening into Right Bronchus.*

1891. Man, aged 58. Dyspnoea increased; resonance at left apex. Cardiac dullness diminished. Dullness with deficient entry and movement; at right base numerous râles.

Post Mortem.—A saccular aneurysm of the descending aorta, which projected to the right, and opened by an aperture, ¼ in. across, into the right bronchus. In the right lower lobe there was marked bronchiectasis. The right upper lobe contained a good deal of aspirated blood, but there was none in the lower lobes.

ANEURYSMS OPENING INTO LEFT BRONCHUS.

CASE XXVII.—*Aneurysm opening into Left Bronchus.*

1908. Man, aged 65, brought in dead. A saccular aneurysm of the arch had compressed and opened into the left bronchus at its origin. The lungs were *bulky and emphysematous.*

CASE XXVIII.—*Aneurysm of descending Aorta opening into Left Bronchus: Tubes in lower lobe dilated.*

1901. Man, aged 56. An impaired note at the left axilla and below the scapula, diminished breath sounds in front, absent behind, dysphagia, pain in back, fatal haemoptysis.

Post Mortem.—A fusiform aneurysmal dilatation at the origin of the descending aorta pressed on and had opened into the left bronchus. The right lung was oedematous. The left lower lobe was fibroid and pneumonic, the tubes were dilated and full of pus.

CASE XXIX.—*Aneurysm of Transverse Aorta eroding into Left Bronchus: Bronchiectasis.*

1901. Man, aged 49. There had been cough for six weeks, with dyspnoea and pain over the left side, beneath scapula especially. The left chest was *larger than the right*. There were distended veins, and there was some tracheal tugging. There was a pleuro-pericardial rub from May 6th to May 28th. There was blood at times, with muco-purulent sputum. The lung was dull behind. On June 22nd the skiagram showed the left lung to be opaque. On July 18th no rub was audible, but there was dullness, with bronchial breathing and intermittent pyrexia. Haemoptysis, which proved fatal.

Post Mortem.—The left pleura was thickened, the left lung was solid with chronic pneumonia, the bronchial tubes were dilated and filled with muco-pus. There was a very large aneurysm of the size of a cocoanut over the second and third parts of the aortic arch, compressing the upper lobe of left lung, also pressing on the left bronchus, practically occluding it and eroding into it 3 c.cm. below the point of bifurcation.

CASE XXX.—*Aneurysm of Transverse Aorta rupturing into Lower Branch of Left Bronchus.*

1901. Man, aged 36. Pain in the left chest for six months, haemoptysis on November 6th. Pain in the right chest on December 1st with marked dyspnoea. The patient could not lie on the left side. There was dullness below the sixth rib on the left side. He had been accustomed to heavy gymnastics and cycling.

Post Mortem.—There was an aneurysm 2¼ in. in size on the lower and distal part of the transverse aortic arch compressing, and rupturing into, the left bronchus 1½ in. from the trachea, involving the left recurrent nerve. The left lower lobe was fibroid and adherent. The tubes were large and prominent and the upper lobe was congested. The right lung was emphysematous at the apex.

CASE XXXI.—*Aneurysm eroding Left Bronchus: Bronchiectasis.*

1901. Man, aged 33. An impaired note at the left apex and base, with deficient breathing. On May 4th, haemoptysis, 2 pints, very deficient breathing, and some impaired resonance. On the 15th there were signs of fluid in the left chest, but there was none found on June 7th, when the chest was opened. On the 18th 2 pints of blood exuded from the wound. He died with exhaustion three weeks later.

Post Mortem.—A saccular aneurysm was found at the anterior wall of the third part of the aortic arch, 3 in. across, occluding, and eroding into, the left bronchus, and compressing the lung. There was a fibroid condition of the left lung with commencing necrosis. The tubes were dilated and filled with a greenish muco-pus, one was dilated in the lower lobe into a good-sized cavity, which was hardly separated from the chest wall and through which blood had passed into the operation wound. There were traces of recent pericarditis. The aneurysm had nearly ruptured into the pericardium.

CASE XXXII.—*Aneurysm: Descending Aorta eroding Left Bronchus: Bronchiectasis.*

1907. Man, aged 31. He worked until three weeks before admission into the hospital, although he had cough and dyspnoea. There was abundant foul expectoration; the left chest was shrunken, with deficient movement and tactile vocal fremitus, with dullness and but little air entry. The cardiac impulse was 1½ in. internal to the nipple, the left pleura was thickened to $\frac{1}{16}$ in.

Post Mortem.—The left lung was fibroid and collapsed, recent pleurisy over the right lung, diffuse bronchiectasis of the left lung, which was riddled with cavities lined by dirty grey recent membrane. The lower lobe was destroyed by recent gangrene, and contained irregular cavities, the largest being 2½ in. by 1 in. and crossed by fibrous bands. The left bronchus was flattened and compressed by an aneurysm, the sac forming a teat-like projection into the tube. There was a mass in the left bronchus, which was dark green and necrosing. There was compensatory dilatation with oedema and bronchopneumonia in the right lung, which was half as large again as the left.

One inch below the aortic arch on the anterior aspect of the descending aorta was an aneurysm 1½ in. across, opening by an orifice ¾ in., compressing the left bronchus.

CASE XXXIII.—*Aneurysm opening into Left Bronchus.*

1897. Man, aged 28. There was a fatal haemoptysis due to a saccular aneurysm opening into the left bronchus ¾ in. from the point of bifurcation.

CASE XXXIV.—*Aneurysm of Descending Aorta eroding into Lower Branch of Left Bronchus: Bronchiectasis: Fibroid Pneumonia.*

1902. Man, aged 38. Pain in the left chest. Pneumonia and pleurisy in October, 1901. Cough and wheezing for one year. Since then he has had occasional haemoptysis; the left chest moved badly and both apices were dull. The left chest was resonant, with deficient air entry. Tactile vocal fremitus and voice sounds deficient. The right side was normal, and later on, the left side showed less marked signs. He died with haemoptysis, after an exploratory operation with a needle in two places, with negative results.

Post Mortem.—He had pleurisy over the left chest, both lungs were large, the right being pale and emphysematous and the left lower lobe was dark, congested, and solid, the lung fibroid, the tubes dilated, and filled with muco-pus; recent septic bronchopneumonia. The lower third of the upper lobe was solid with chronic pneumonia. A saccular aneurysm of the transverse and descending aorta had pressed on the left bronchus, especially the branch leading to the lower lobe; it had bulged and eroded into it with fatal results. There was blood clot in the bronchi on both sides.

CASE XXXV.—*Aneurysm opening into Left Bronchus.*

1892. Man, aged 49. There was cough, pain in the left chest, deficient breathing at the left apex in front. Fatal haemoptysis.

Post Mortem.—A diffuse aneurysm of the transverse aorta opening into the left bronchus. Some collapse in the left upper lobe; diffuse haemorrhage into both lungs.

CASE XXXVI.—*Aneurysm of Descending Aorta opening into Left Bronchus and Oesophagus: Tubes at Base Dilated.*

1906. Man, aged 37. Pain over the left scapula, dyspnoea, dullness between the scapulae. Diminished entry of air into the left lung; fatal haemorrhage into the stomach.

At inspection two aneurysms 2½ in. across were found on the descending aorta; one compressed the left bronchus and also opened into the oesophagus. Slightly dilated tubes at the left base. Right lung emphysematous.

ANEURYSMS MERELY COMPRESSING LEFT BRONCHUS.

CASE XXXVII.—*Aneurysm compressing Left Bronchus and Lung: Rupture into Pleura.*

1899. Man, aged 48. For seven months pain in the left side of the neck and for four months in the chest. An aneurysm pulsated in the first and second left spaces, pressing on the left recurrent laryngeal nerves.

Post Mortem.—It arose from the transverse arch, and had compressed the trachea and the left bronchus. There was no bronchiectasis, because the left lung was also compressed. There were eight pints of blood in the left pleural cavity, into which it had finally ruptured.

CASE XXXVIII.—*Aneurysm pressing on Left Bronchus.*

1904. Man, aged 43. The left chest was resonant; there was no cardiac dullness; deficient entry of air into the left chest; cough, haemoptysis, pain for four months; dyspnoea, abundant sputum. He was said to have bronchiectasis on the left side. The skiagram on March 2nd, 1903, shows nothing abnormal. Absence of breath sounds at the left base; (?) aneurysm pressing on the left bronchus. A skiagram taken on October 23rd, 1903, shows the heart was displaced to the right. November, 1903, râles and rhonchi in both chests, with increasing deficiency of breath sounds on the left side, but it is if anything more *resonant* than the right. December 12th, 1903, a skiagram showed a clear chest with no signs of aneurysm or growth. The left pulse was smaller and was delayed; good note on the left side in front; cardiac dullness obliterated; breath sounds feeble.

CASE XXXIX.—*Aneurysm compressing Left Bronchus: Septic Bronchopneumonia: Early Bronchiectasis.*

1901. Man, aged 46 Pain for two years in the upper dorsal region with orthopnoea. An aneurysm behind the sternum; deficient entry of air in the left lung.

Post Mortem—An aneurysm of the second and third parts of the aortic arch had pressed on the manubrium, the left bronchus and recurrent nerve, also on the left lung, but had not opened into the bronchus. There was a septic bronchopneumonia of the left lung, especially at the base, which had broken down in places. The tubes were slightly dilated with foul mucopus; the left upper lobe was oedematous.

CASE XL.—*Aneurysm compressing Left Bronchus.*

1888. Man, aged 63. Two years' dyspnoea and pain; dysphagia one month; hyper-resonant note over axilla and base; deficient entry of air into the left base.

Post Mortem.—The right lung was small and emphysematous: the left lung was markedly collapsed and airless over the base; the left bronchus was compressed by a large aneurysm of the descending aorta, which also compressed the left lower lobe.

CASE XLI.—*Aneurysm of Descending Aorta compressing Left Bronchus: Large Intercommunicating Cavities in Left Lung.*

1906. Man, aged 31. Cough with pain in the left side, deficient entry of air at the left apex. On August 22nd there were no breath sounds audible on the left side; he sweated profusely, and had a cough. January 24th, 1906, there was a pleuritic rub; the pain in the left side was more severe. He was explored twice for fluid, but only blood was obtained. A pulsating swelling was detected behind on February 2nd.

Post Mortem.—For 3 in. beyond the aortic arch there was a 4 in. sac about the size of a cocoanut. The left pleura was thickened and adherent. An aneurysm had compressed the left lung and bronchus, which latter was flattened and atrophied. The left lung had been converted into a multilocular sac, containing 4½ pints of foul pus. There were a series of intercommunicating cavities with fibrous septa composed of the remains of the lung tissue,

CASE XLII.—*Aneurysm of Descending Aorta compressing the Left Bronchus: Bronchiectasis Left Base.*

1908. Man, aged 33. Haemoptysis 1907. The left pupil was small; there was dysphagia, pain on the left side, impaired note at the left base, a pleuritic rub at both bases.

Post Mortem—There was a saccular aneurysm of the transverse and descending aorta which had during life probably compressed the left bronchus and caused the local bronchiectasis, but did not do so appreciably *post mortem*, when it was flaccid. The left base was solid, the

left lower lobe was fibroid, the bronchi were dilated with thickened and red mucous membrane.

CASE XLIII.—*Aneurysm compressing Left Bronchus: opening into Pulmonary Artery.*

1902. Woman, aged 51. Pain in the left shoulder for three months. Cough one year. Impaired resonance in the first and third left spaces, with deficient entry of air. Fatal cyanosis and dyspnoea. Paralysis of left recurrent nerve due to an aneurysm.

Post Mortem.—The left lung, especially the middle and lower parts, were collapsed from the pressure of the aneurysm; there had been pressure on the left bronchus, from a saccular aneurysm of the ascending aortic arch, which finally ruptured into the pulmonary artery.

CASE XLIV.—*Aneurysm pressing on Left Bronchus.*

1888. Man, aged 57. Acute pneumonia of the lower lobes; deficient breathing and voice sounds over the left chest, with deficient movement. No dullness. Aortic incompetence.

Post Mortem.—A diffuse atheroma in the ascending and transverse arch of the aorta, with a saccular aneurysm 1½ in. across pressing on the trachea and on the anterior surface of the left bronchus. Acute pleuropneumonia in both lower lobes.

CASES OF EPITHELIOMA OF OESOPHAGUS AFFECTING THE BRONCHI.

CASE XLV.—*Epithelioma of Oesophagus, Secondary Deposit in Gland, compressing Lower Branch of Left Bronchus: Bronchiectasis.*

1901. Woman, aged 36. Dullness at the left apex, both back and front, deficient breathing at the left apex, general râles and rhonchi, persistent cough, breathing somewhat stridulous. Gland at the episternal notch pressing on the trachea.

Post Mortem.—An epithelioma of the oesophagus, with secondary deposits in the lungs. The upper lobe was nearly solid with growth; there was some deposit in the lower lobe with bronchiectasis, the left lower bronchus being partially constricted by the pressure of an enlarged gland.

CASE XLVI.—*Epithelioma of Oesophagus opening into Left Bronchus: Septic Bronchopneumonia.*

1892. Man, aged 50. Epithelioma of the oesophagus opening into, by an aperture ¾ in. by ½ in., the left bronchus. Some septic bronchopneumonia, especially in the left lung.

CASE XLVII.—*Epithelioma of Oesophagus opening into Left Bronchus.*

1894. Man, aged 51. There was an epithelioma of the oesophagus opening into the left bronchus, with diffuse bronchopneumonia, but most marked on the right side.

CASE XLVIII.—*Epithelioma of Oesophagus invading Left Bronchus: Septic Bronchopneumonia.*

1900. Man, aged 45. There was a history of vomiting for four months. Dysphagia, gastrostomy. He improved greatly until February 12th, when there was cough with expectoration. On the 20th the right base was dull; breath sounds were deficient. At the left base there was bronchophony and pectoriloquy.

Post Mortem.—An epitheliomatous growth was found in the oesophagus, invading the left bronchus, and there was a diffuse septic bronchopneumonia.

CASE XLIX.—*Epithelioma of the Oesophagus eroding the Right Bronchus: Gangrene.*

1899 Man, aged 58. Bronchial breathing, with râles over the right lower lobe behind. Pyrexia; no dullness; foul breath.

Post Mortem.—An epithelioma of the oesophagus invaded the right bronchus and had set up a gangrenous cavity 2 in. across in the right lower lobe.

CASE L.—*Growth compressing Right Bronchus: Pleuritic Effusion: Pneumonia.*

1900. Man, aged 51. He was ill for six months; there was deficient breathing, no voice sounds, with oedema of the right chest, secondary growths in the glands and skin.

Post Mortem—Pleuritic effusion, with growth at the root of the lung. The right bronchus ran into a mass of growth; the tubes were filled with pus. There was pneumonia at the edge of the lung.

CASE LI—*Epithelioma of Oesophagus eroding Right Bronchus: Gangrene: Septic Bronchopneumonia.*

1904. Man, aged 39. Dullness over the right chest with deficient entry of air and crackling râles.

Post Mortem—Epithelioma of the oesophagus, which was sloughing and opening into the right bronchus. There was a gangrenous cavity in the right upper lobe and a septic bronchopneumonia below.

CASE LII—*Epithelioma of Oesophagus opening into Right Bronchus: Abscess Cavities in Lung.*

1905. Man, aged 62. A history of dysphagia for eighteen months, with offensive breath and dyspnoea.

Post Mortem—An epithelioma of the oesophagus opening into the right bronchus. There were abscess cavities in the right middle lobe with bronchopneumonta at the base.

CASE LIII.—*Epithelioma of Oesophagus invading Right Bronchus: Collapse of Lung beyond.*

1899. Man, aged 52. A duodenostomy was performed for an epithelioma of the oesophagus.

Post Mortem.—The right bronchus was also invaded by growth, and the middle and lower branches were occluded. There was bronchopneumonia of the right upper lobe, and the middle and lower lobes were airless and collapsed.

CASE LIV.—*Epithelioma of Oesophagus pressing on Left Bronchus.*

1898 Man, aged 58. Admitted for dysphagia with hoarseness. The left vocal cord was paralysed.

Post Mortem.—The root of the right lung was invaded by growth. As a result the left bronchus was stenosed. Both bases were oedematous. There was an epithelioma of the oesophagus.

CASE LV.—*Epithelioma of Oesophagus opening into Left Bronchus: Foul Abscesses in Lung.*

1889. Man, aged 50. Epithelioma of the oesophagus opening into the left bronchus. There were numerous foul acute abscesses in the base behind. The right lung was free.

CASE LVI.—*Epithelioma of Oesophagus opening into Left Bronchus: Gangrene of Base of Lung.*

1882. Man, aged 51. Five months' dysphagia, cough. Deficient resonance with diffuse bronchial breathing and bronchophony over the right base. Foul breath.

Post Mortem.—Epithelioma of oesophagus opening into the left bronchus. Pneumonia with commencing gangrene at the base of the right lung.

CASE LVII—*Epithelioma of Oesophagus opening into Right Bronchus.*

1891. Woman, aged 35.

There was epithelioma of the oesophagus opening into the right bronchus, with deficient entry of air into the right lung. Septic bronchopneumonia of the right lung; the base was gangrenous, and there was a large opening from the oesophagus into it.

CASE LVIII.—*Epithelioma of Oesophagus opening into Left Bronchus.*

1888. Man, aged 65. Three months' dysphagia. Vomiting, wasting, cough with expectoration; later dyspnoea, râles over chest.

Epithelioma of the oesophagus, which opened into the left bronchus by a hole admitting the tip of the finger, and some nodules were fungating into the right bronchus. There was bronchopneumonia with oedema of the lungs.

CASES OF SARCOMA OF BRONCHIAL GLANDS COMPRESSING THE BRONCHI.

CASE LIX.—*Sarcoma compressing especially Left Bronchus and Pulmonary Artery: Gangrene.*

1889. Man, aged 60.

Post Mortem.—A mediastinal sarcoma surrounded the bronchi and invaded the left one, almost occluding it. The right was slightly compressed. The left pulmonary artery was extremely reduced in size by the growth around it; there was acute gangrene of the upper part of the left upper lobe, the base was airless, the right lung was normal; there was no dilatation of the tubes.

CASE LX.—*Sarcoma compressing the Left Bronchus: Necrosis of Lung: Left Pneumothorax.*

1905. Man, aged 63. Cavity at the apex. There was dysphagia, hoarseness, and the left vocal cord was paralysed. There was dullness at the left apex with deficient breath sounds.

Post Mortem.—There was sarcoma of the glands in the mediastinum extending into the root of the left lung, and obstructing the left bronchus. There were traces of old tubercle at the left apex, with a recent cavity due to extension of the growth and necrosis, leading to pneumothorax.

CASE LXI.—*Sarcoma compressing Right Bronchus: Pleuritic Effusion: Oedema of Lung.*

1895. Man, aged 36. Admitted with pain, dullness at the right base, with deficient entry of air; oedema of the back. A rub could be heard, and there was dyspnoea; 8 pints of fluid were drawn off.

Post Mortem.—Sarcoma of the glands, surrounding and pressing on the right bronchus, not occluding it, with invasion of the right upper lobe at the root. The rest of the lung was solid with bronchopneumonia. The left lung was oedematous.

CASE LXII.—*Sarcoma invading Left Bronchus: Gangrene of Lung: Cavitation.*

1899. Woman, aged 22. She was ill for three months with pain in the left chest, which was dull; there was no tactile vocal fremitus, voice or breath sounds. On the 18th the dyspnoea was intense, and 21 oz. of turbid fluid were taken from the left chest. On the 19th, 4 oz. were drawn from the pericardium. The chest was very tender. On the 23rd, 3 oz. of pus were removed. The patient died while an attempt was being made to drain the pleura.

Post Mortem.—A sarcomatous growth was found, invading the left bronchus at the bifurcation. There was a necrotic bronchopneumonia, especially at the lower lobe, which contained a quantity of pus in numerous large cavities. Lymph was found over the pleura.

CASE LXIII.—*Growth in Glands compressing Left Bronchus: Gangrenous Cavity.*

1901. Man, aged 46. Pain in the chest with paraplegia, defective movement of the left chest with crepitations in the axilla.

Post Mortem.—The left bronchus was invaded by a growth commencing in the mediastinum. The upper lobe was collapsed at the apex and contained a gangrenous, ragged cavity, 3 in. across. Some growth in the upper lobe. The lower lobe was pneumonic.

CASE LXIV.—*Growth at Root of Right Lung compressing Bronchus: Bronchiectasis.*

1904. Woman, aged 43. There was a history of chest symptoms for two years. The right chest was dull, with deficient breath and voice sounds; 23 oz. of serum were drawn off. There was dyspnoea, which was fatal the day after admission.

Post Mortem.—A growth in the mediastinum, 3 in. across, surrounded the right bronchus and reduced its lumen to one fourth; it had also invaded the lung. The pleura was firmly adherent to the chest wall. The right lung was fibroid and compressed, and there was some dilatation of tubes in the lower lobe. Bronchopneumonia.

CASE LXV.—*Growth compressing Right Bronchus: Bronchiectasis: Gangrene.*

1898. Man, aged 57. He had had dyspnoea for four months, with stridor and haemoptysis for one month. The right chest was dull. There was cavernous breathing behind down to the spine of the scapula, and deficient breathing below.

Post Mortem.—The right lung was invaded by growth, breaking down in places and also compressing the bronchus. The few tubes which remained in the right upper lobe were dilated; the left lung was oedematous.

CASE LXVI.—*Sarcoma compressing Left Bronchus and invading Lung.*

1902. Woman, aged 20. She had a cough and there were large glands in the neck. The left chest was dull; there was no bronchial breathing and no dysphagia.

Post Mortem.—There was a sarcomatous mass in the posterior mediastinum invading the left lung, which it almost entirely occupied.

CASE LXVII.—*Sarcoma compressing Upper Branch of Left Bronchus: Infarct in Upper Lobe.*

1905. Woman, aged 59. There were dyspnoea, cyanosis, orthopnoea. The left chest was dull except at the base, where the resonance was only deficient. There were no breath sounds over this side; 90 oz. of fluid were drawn from the left side.

Post Mortem.—There was a mass of glands behind the trachea and compressing the upper branch of the left bronchus. The left lower lobe was solid with septic bronchopneumonia, and there was an infarct in the upper lobe.

CASE LXVIII.—*Sarcoma compressing Right Bronchus: Bronchiectasis.*

1905. Woman, aged 57. There was a cough, with *stridor* and increasing dyspnoea.

Post Mortem.—There was a mass of growth in the right lung at the root, surrounding the bronchus. The rest of the lung was solid. The bronchial tubes in the lower part were dilated. The tubes were full of muco-pus. A mass of round-cell sarcomatous growth invaded the anterior surface of the trachea.

CASE LXIX.—*Sarcoma compressing Right Bronchus.*

1905. Woman, aged 54. There was cough and wasting. There were dilated veins on the right side of the chest. The gland behind the right sterno mastoid was invaded by growth. Dullness at the right apex.

Post Mortem.—A large mass of sarcomatous growth in the anterior part of the mediastinum had invaded the lung at the right apex and compressed the bronchus. It had also invaded the walls of the innominate veins.

CASE LXX.—*Sarcoma compressing Right Bronchus: Pleuritic Effusion.*

1905. Woman, aged 19. There was a sarcoma of the humerus, rapidly invading the glands and the thorax.

Post Mortem.—There were 30 oz. of fluid in each chest. The growth penetrated the trachea just above the bifurcation, and compressed the right bronchus and superior vena cava. There were masses of growth in the anterior and posterior mediastinum, and nodules of growth in the right lung. There was recent lymph on the surfaces. There was an entire absence of any dilatation of the bronchi owing to the fluid in the chest.

CASE LXXI.—*Sarcoma invading Right Bronchus: Bronchiectasis: Gangrene.*

1902. Man, aged 50. He had had a cough for two months, and there was dyspnoea.

Post Mortem.—There was sarcoma of the glands at the root of the right lung; the right bronchus was invaded and eroded on its anterior surface. The tubes of the right lower lobe were dilated, and there were gangrenous cavities.

CASE LXXII.—*Occlusion of Left Bronchus by Growth in Glands.*

1905. Woman, aged 39. There was dyspnoea for nine months and haemoptysis lasting one week. There was cyanosis; the left chest was dull in front; the note was good at the left scapula, but dull elsewhere. There was a good entry of air over the right lung; the heart impulse was outside the nipple.

Post Mortem.—There was a mass of growth at the root of the left lung, extending along the bronchus into the lung, which it largely infiltrated. There was complete blocking of the bronchus for 2 in. from the place of origin.

CASE LXXIII.—*Right Bronchus compressed by Growth: Effusion into Pleura.*

1906. Male, aged 8. A right pleural effusion, which was tapped several times. There was dullness at the base.

Post Mortem.—The right bronchus was compressed by growth, and most of the upper lobe was filled with growth. There was bronchopneumonia in the right middle and lower lobes, which were airless, solid, and had been previously compressed by fluid. There was now lymph on the surface.

CASE LXXIV.—*Sarcoma invading Right Bronchus: Pleuritic Effusion: No Bronchiectasis.*

1902. Man, aged 40. There were cough, dyspnoea, and pain; haemoptysis for three weeks. He was cyanosed; the right chest bulged; there was deficient resonance at the apex, with râles; the rest of the right chest was dull; 102 oz. of blood-stained fluid were drawn from the right chest.

Post Mortem.—There was a sarcomatous growth in the glands; the right bronchus was compressed. Tne root of the right lung was infiltrated with growth, the tubes of the lower part being filled with pus, but not dilated. The lung was also compressed by 60 oz. of fluid.

CASE LXXV.—*Growth compressing Left Bronchus: Pleuritic Effusion: Gangrene.*

1893. Man, aged 35. Haemoptysis, dysphagia, dullness over the left chest. Dry tapping. There was growth in the gland behind the left sterno-mastoid.

Post Mortem.—A mass of growth in the mediastinum extended round the left bronchus, and occluded it. The left upper lobe was collapsed; the left base was compressed by fluid. There were some nodules of growth in the lung. A patch of gangrene in the upper lobe.

CASE LXXVI.—*Primary Sarcoma of Glands compressing the Right Bronchus.*

1888. Man, aged 36. There had been dyspnoea for six weeks. The right chest was dull for the first three spaces. Harsh breathing at the right apex, with rhonchi. There was some bulging of the right chest, with oedema. The chest was explored for fluid, but with negative results. There was harsh breathing over the left lung.

Post Mortem.—A widespread sarcoma had invaded the lung, and at the left base a mass had broken down into a cavity about 2 in. across. The lower branches of the right bronchi were occluded by growth. There was no dilatation of tubes. There was growth along the aorta as far as the sacrum.

CASE LXXVII.—*Primary Growth in Mediastinum compressing Right Bronchus.*

1888. Woman, aged 67. Carcinoma of the mediastinum. The right chest moved less than the left, there was deficient entry of air, but the resonance was normal; later some dullness at the root.

Post Mortem.—The right bronchus was constricted, but not invaded, by a growth, and would only admit a probe. The tubes beyond were slightly dilated, and contained muco-pus.

CASE LXXVIII.—*Growth at Root of Right Lung compressing Bronchus.*

1888. Man, aged 84. The right sterno-mastoid gland was enlarged, the right chest resonant, no entry of air. One month later the right chest had bulged at the upper part.

CASE LXXIX.—*Growth invading Left Bronchus.*

1907. Woman, aged 66. Cough, pain, fluid in the left chest. Oedema of the left chest with defective movement. There was a faint bruit at the apex, but none below. Tactile vocal fremitus was faint, there was dullness of the apex in front, and all over behind. Heart dullness ½ in. to the right. Dyspnoea. Blood in the sputum.

Post Mortem.—A mass of growth at the root of the lung growing into the left lower lobe and into the left bronchus. The growth also invaded the upper lobe.

CASE LXXX.—*Growth at Root of Right Lung.*

1907. Man aged 62. A cough for fourteen months. A mass of glands in the right side of the neck for two months. Oedema of the right chest wall, slight stridor, growth in the pancreas. Nodule in the skin. No deficient resonance over the right chest, yet in a skiagram an opacity due to a growth was visible at the root of the lung. Five days before death there was dullness in the first space. Increasing fatal dyspnoea.

CASE LXXXI.—*Cancerous Glands compressing the Left Bronchus.*

1900. Man, aged 42. Dysphagia and paresis of the left recurrent laryngeal nerve, deficient breathing at the left apex, some haemoptysis.

Post Mortem.—Cancer of the pancreas, secondary deposit in the mediastinum, compressing the oesophagus, the left bronchus and the root of the lung. No ulceration. Lung oedematous.

CASE LXXXII.—*Lymphadenoma (Sarcoma?) compressing and eroding into Right Bronchus: Pleuritic Effusion.*

Man, aged 24. Admitted with swollen oedematous face with general enlargement of glands. Deficient movement and entry of air with bronchophony and dullness over the left lung. Left pleuritic effusion; nothing abnormal about breath sounds on the right.

Post Mortem.—"Lymphadenomatous" growths in the cervical and thoracic glands; infiltration of root of the right lung; one mass had eroded into the right bronchus an inch from its origin; the growth extended freely into the lung; 175 oz. of fluid in the left chest.

CASE LXXXIII.—*Lymphadenoma (? Sarcoma) of Glands fungating into Bronchus: Bronchiectasis of Upper Lobe: Empyema.*

1888. Man, aged 48. Admitted with diagnosis of lymphadenoma. Large tonsils; haemoptysis six months ago; deficient movement and breath sounds over the left chest. Pleuritic effusion, tapped in March. Empyema drained in April. Death resulted from haemorrhage while on the operating table.

Post Mortem.—There was a universally distributed "lymphadenoma" on the left side of the thorax; three encysted empyemata; the left lung was carnified with an excess of fat. Bronchiectasis of the right upper lobe, with cavities ¾ to ½ in. corresponding to bronchus, which had been compressed by a gland which had at one point fungated into the bronchus. The right lung was emphysematous.

SECONDARY GROWTHS IN BRONCHIAL GLANDS AFFECTING THE BRONCHI.

CASE LXXXIV.—*Secondary Growth invading the Right Bronchus: Bronchiectasis.*

1902. Man admitted with deficient movement of the right chest, which was absolutely dull both back and front. Diminished tactile vocal fremitus. Bronchial breathing at the apex, which was heard faintly down to the eighth rib, but none below. (?) Paralysis of the right sixth nerve.

Post Mortem.—Primary growth in the parotid; a secondary deposit infiltrated the root of the right lung, and surrounded the right bronchus, which was ulcerated, and ½ in. from its origin the lumen was reduced to the size of a crow-quill. Beyond there was a sac of dilated bronchioles with foul contents. There were multiple red patches on the dura mater indicative of growth.

CASE LXXXV.—*Secondary Growth invading Right Bronchus*

1900. Man, aged 26. Admitted with expectoration sometimes of blood. There was oedema of the right side of the chest with dilated veins, with diminution of resonance and of voice and breath sounds, on the right side.

Post Mortem.—Sarcoma of the head of the pancreas; secondary growth in the right upper lung, invading the right bronchus and right auricle.

CASE LXXXVI.—*Secondary Growth in Glands compressing Left Bronchus: Upper Tubes Dilated: Lower Gangrenous.*

1907. Man, aged 59. One year pain in the back. Four years ago he had a severe fall, which jarred his coccyx. There was diminished entry of air at the right base; above it was good; there was some dullness below and an impaired note above.

Post Mortem.—Primary sarcoma of lumbar spines. Diffuse secondary deposits at the point of bifurcation of the trachea, which also compressed the left bronchus; the left upper tubes were dilated. The lower part of the left lung had a large cavity with growth in its wall. Septic bronchopneumonia.

CASE LXXXVII.—*Secondary Growth in Glands compressing Right Bronchus.*

1888. Female aged 30. There was a cystic sarcoma of the breast with recurrences, one of which was found *post mortem* to have compressed and invaded the right bronchus. The lung was normal.

CASE LXXXVIII.—*Secondary Growth in Glands compressing Left Bronchus.*

1889. Woman, aged 36. There was a medullary carcinoma of the ovary, with extensive secondary deposits. The lower one-third of the left upper lobe was solid with infarction; the left bronchus was surrounded and compressed by growth, and the rest of the lung was airless collapsed, and compressed by 10 oz. of fluid. Some collapse at the right base.

CASE LXXXIX.—*Left Bronchus compressed by a secondary Epitheliomatous Gland: Bronchiectasis.*

1901. Woman, aged 56. She was admitted with bronchitis. There was dullness at the left apex, with deficient breath and voice sounds. Dyspnoea, cough, and stridor. There was a gland above the sternum.

Post Mortem.—Epithelioma of the oesophagus; the left bronchus was compressed by a gland. Disseminated growths in the left lung; growths at the root of the lung, with bronchiectasis.

CASEOUS GLANDS COMPRESSING OR OPENING INTO BRONCHI.

CASE XC.—*Left Bronchus compressed by a Caseous Gland Cavity in Lower Lobe*

1898. Boy, aged 4. The left chest was dull with deficient entry of air, but the results of an exploratory operation were negative.

Post Mortem.—The left bronchus was compressed by a caseous gland; there was a cavity $\frac{3}{4}$ in. across in the lower lobe. There was a caseous bronchopneumonia. A gland near the branch of the lower bronchus had softened. Phthisis.

CASE XCI.—*Caseous Gland pressing on Right Bronchus.*

1902. Male, aged 4. For six weeks cough with dyspnoea; the movements of the right chest were defective, and there was deficient breathing, especially at the right apex. The breathing on the left side was loud and harsh; voice sounds deficient. July 2nd: Resonance at the apex was tympanitic; there was an impaired note, with deficient breath sounds at the right base. Bronchial breathing at the right apex, the lung slowly becoming fibroid, probably due to a caseous gland pressing on the right bronchus.

CASE XCII.—*Gland compressing Right Bronchus.*

1907. Female, aged 8. Cough for one month. Some dullness of the left chest in front. Impaired note behind. In the right chest there was some dullness below, and the note was impaired above. Skiagram shows deficient movement on the right side, with an opaque area at the root. Consonating râles at the right base.

Post Mortem.—General caseous bronchopneumonia, with a gland pressing on the right bronchus.

CASE XCIII.—*Glands occluding Right Bronchus.*

1902. Boy, aged 8 months. Admitted moribund, supposed to be diphtheritic.

Post Mortem.—Caseous bronchial glands completely occluding the bronchus up to the right middle lobe. The right middle lobe was airless and full of muco-pus. There was no similar condition in the rest of the lung. General tuberculosis.

CASE XCIV.—*Gland compressing Right Bronchus.*

1904. Child, aged 1. There was an impaired note at the left apex and over the sternum, with râles and deficient entry of air.

Post Mortem.—General tuberculous bronchopneumonia, with a gland compressing the root of the right bronchus.

CASE XCV.—*Right Bronchus compressed by Gland.*

1902. Boy, aged 5. Measles, general râles in the chest, some dullness at the right base, bronchial breathing in the right axilla.

Post Mortem.—The bronchus to the right middle lobe was occluded by the pressure of a caseous gland 2½ by 1 in., the lobe was airless. There was collapse with bronchopneumonia and recent lymph on the surface, with diffuse tuberculosis.

CASE XCVI.—*Caseous Gland opening into Right Bronchus.*

1899. Boy, aged 3. Tuberculous meningitis.

Post Mortem.—A large caseous gland had opened into the right bronchus; nodules of tubercle were scattered about the lung.

CASE XCVII.—*Phthisis: Glands opening into Right Bronchus and also into Oesophagus.*

1895. Woman, aged 76. For two months she had had bronchitis with haemoptysis, which recurred and proved fatal. There was pain, with some dullness over the right side. Two ounces of pus drawn off from pleura.

Post Mortem.—Phthisis and chronic bronchopneumonia. A caseous gland had softened and opened into the oesophagus and also into the right bronchus.

CASE XCVIII.—*Phthisis: Caseous Gland opening into the Right Bronchus.*

1901. Man, aged 33. There was dullness at the right base and an impaired note at the left apex with a rub at the left axilla. He died with acute miliary tubercle.

Post Mortem.—A healed phthisis at the left apex. A caseous gland had opened into the right bronchus.

CASE XCIX.—*Suppurative Pylephlebitis: Abscess in Bronchial Glands opening into the Right Bronchus: Secondary abscesses in Brain.*

1889. A man, aged 23, was admitted with rigors, vomiting, and pain in the hepatic region. He had a series of epileptiform attacks with an erratic pyrexia and died pyaemic and comatose.

Post Mortem.—There was suppurative pylephlebitis with abscesses in the spleen and brain and general meningitis. There was also an abscess in a bronchial gland which contained half a drachm of pus, and had opened into the right bronchus near the bifurcation. The larger bronchi contained mucopus.

CASE C.—*Suppurating Gland opening into Right Bronchus.*

1889. Man, aged 23. No special signs noted in lung.

Post Mortem.—A suppurative pylephlebitis, with cerebral abscess. A suppurating bronchial gland had opened into the right bronchus.

CASE CI.—*Suppurating Gland opening into Both Bronchi.*

1889. Man, aged 19. Caries of atlas.

Post Mortem.—A softened caseous gland at bifurcation of the trachea had opened into each bronchus.

ABSCESSES OPENING INTO BRONCHI.

CASE CII.—*Empyema bursting into Right Bronchus.*

1901. Man aged 65. Alcoholic and syphilitic. There was some dullness at the right base up to the sixth rib, with crackling râles and diminished voice sounds. He had been ill for six weeks. Sudden expectoration of foul pus; death two days later.

Post Mortem.—A foul empyema of 17 oz. had burst into the right bronchus. There was an old scarred liver and two stones were found in the gall bladder.

CASE CIII —*An Abscess opening into the Oesophagus and also into the Right Bronchus, producing Gangrene of the Lung.*

1893. Male, aged 12. He was ill for four days, with fetid breath, pain on the right side, cough, vomiting, and diarrhoea. Foul blood and muco-pus was expectorated. There was dullness, with bronchial breathing, bronchophony, and consonating râles, over the left scapula. An attempt was made to drain the gangrenous cavity, without benefit.

At the *inspection* an abscess was found, which had opened into the right bronchus, and so had produced a large acute gangrenous cavity in the right lung. It also had communicated with the oesophagus. The origin of the process remained obscure.

GUMMATA PRESSING ON BRONCHI.

CASE CIV.—*Gumma pressing on Right Bronchus: Upper Lobes Over-distended: Fluid at Base.*

1889. Man, aged 64. Intense paroxysmal dyspnoea for ten months; dilated veins over the chest. Prolonged expiration. Dullness behind sternum; deficient entry of air on the right side. He died twenty-four hours after admission.

Post Mortem.—The right bronchus in the upper division was obstructed by a gummatous mass; the upper and middle lobes were over-distended, the lower were airless from compression by 5 pints of fluid; the left lung was dry and emphysematous.

CASE CV.—*Gumma compressing Right Bronchus: Bronchiectasis: Pleuritic Effusion.*

1890. Woman, aged 46. Oedema of the face and chest with cyanosis.

Post Mortem.—A gummatous mass in the chest which compressed the superior vena cava and the right bronchus, and the branches beyond were uniformly dilated up to their extremity and their walls thickened. The lung was solid. Pleuritic effusion on the left side.

SYPHILITIC STENOSIS OF BRONCHI.

CASE CVI.—*Syphilitic Stenosis of the Right Bronchus.*

Man, aged 42. Impaired note over the right chest with deficient entry of air. Râles at the base. Slight hemiplegia.

CASE CVII.—*Syphilitic Stenosis of Bronchi: Bronchiectasis.*

1901. Man, aged 36. Deficient respiration on the left side with stridor on the right. The chest was *hyperresonant*, the cardiac dullness was diminished. A rub was audible at the left base; the temperature was 104°. Later, in February, dullness with bronchial breathing was audible over left chest and a rub in the axilla and over the front. He was venesected.

Post Mortem.—Syphilitic stenosis of both bronchi, the left admitted a quill pen, but the right was larger. The walls of the trachea and bronchi were surrounded by thickened tissue. The bronchioles were not thickened; the bronchioles at the left lower lobe were all dilated. There was a recent grey hepatization of the left lung, with thick muco-pus in the bronchi, less marked dilatation of the tubes on the right side.

CASE CVIII —*Syphilitic Stenosis of the Larynx, Trachea and Right Bronchus: Bronchiectasis.*

1905. Male, aged 39. Has been in the navy. Has had a sore throat for two years. Haemoptysis a year ago, with aphonia, which has been persistent for the past year. No vesicular murmur on the left side of the chest, the arytenoids are greatly thickened, and the cords do not move. Dysphagia.

Post Mortem.—Syphilitic stenosis of the trachea, larynx, and of the right bronchus near its origin. Bronchiectasis on both sides, with fibroid thickening. Gummata in the liver.

CASE CIX.—*Phthisis: Syphilitic Stenosis of Left Bronchus: Bronchiectasis.*

1889. Woman, aged 32. She has been ill for ten years with expectoration and wasting. The left chest was dull, with defective movement, no tactile vocal fremitus. Right apex deficient, resonance in front, normal behind. Bronchial and later cavernous breathing at left apex, no breath sounds below.

Post Mortem.—There was phthisis at the right apex. There was a scar in the trachea at the point of bifurcation and stenosis at the orifice of the left bronchus, which only admitted a No. 3 catheter. A very large cavity at the left apex, lung fibroid with great bronchiectasis. There was a small cavity at the apex of the right lung and a few tubercles. Perforation of soft palate.

CASE CX.—*Syphilitic Ulceration of Trachea: Stenosis of Both Bronchi: Early Bronchiectasis.*

1893. Man aged 29. Syphilis seven and half years ago. Cough two months. Dyspnoea on exertion. He was admitted into the hospital with extreme dyspnoea. Tracheotomy gave no relief.

Post Mortem.—There was an ulcer on the lower end of the trachea, with stenosis of both bronchi and thickening of the walls. The right would just admit a probe, and the left the tip of the finger. The tubes beyond were thickened and dilated. There was a recent bronchopneumonia of the right base, and lymph over the left lung.

PRIMARY CANCER OF BRONCHUS.

CASE CXI.—*Primary Cancer of Left Bronchus: Pleuritic Effusion.*

1899. Man, aged 58. He had had dyspnoea and bronchitis for four months, with distended veins on the chest for three months. Dysphagia and oedema of arms for two months. The right chest was dull, with absence of breath sounds below the fourth rib. Orthopnoea; 56 oz. of foul fluid were drawn from the left chest.

Post Mortem.—The upper lobe was gangrenous, with a large cavity, the lower compressed by fluid. A large mass of columnar-cell carcinoma was present in the mediastinum, compressing the left bronchus and veins, and ulcerating into the oesophagus. The growth had invaded both the left bronchus and the trachea.

CASE CXII.—*Primary Cancer of Right Bronchus: Pleuritic Effusion: Bronchiectasis.*

1899. Man, aged 52. Admitted with purpura and septicaemia and great weakness; deficient breath sounds with deficient movement and dullness over the right side; tactile vocal fremitus normal; no dyspnoea. His illness commenced with pleuritic effusion eighteen months ago, and he has never been well since. Swelling of the thigh followed in a few days by a fractured femur.

Post Mortem.—Primary carcinoma of right bronchus, with a secondary deposit in the fractured femur, heart, and kidneys. The right lower bronchus was nearly occluded

by a growth which had also invaded the lung; the pleura in this part was ¾ in. thick and fibroid. There was bronchiectasis; the tubes were filled with foul greenish pus, with patchy necrosis of the right lung.

CASE CXIII.—*Primary Cancer of Right Bronchus: Gangrene at Base: Pleuritic Effusion.*

1899. Woman, aged 52. She had a right pleurisy due to malignant disease, which was tapped three times and finally drained.

Post Mortem.—There was an enlarged kidney, with an extensive, dense growth in the right lung and pleura. The right lung was compressed to one-eighth of the normal size; the pleura was much thickened. The lung was solid, compressed, and airless, with a small gangrenous cavity at the base. The main bronchi were thickened; the tubes were not dilated. There were secondary deposits in the right kidney. The growth was a primary columnar-cell carcinoma of the right bronchus.

CASE CXIV.—*Primary Cancer of Left Bronchus.*

1899. Man. Admitted with right facial paralysis, with three nodules of growth on the head and several in the abdominal wall. Dullness at the right base below the angle of the left scapula. Diminished entry of air; tactile vocal fremitus normal.

Post Mortem.—The right lung was extensively infiltrated with growth, and there were disseminated nodules over the left lung. Diffuse bronchopneumonia. Primary columnar-cell carcinoma of the left bronchus, with diffuse growth and bronchopneumonia in the left lung. There were secondary deposits in the skin, scalp, right sterno-mastoid gland, and liver.

CASE CXV.—*Cancer of Left Bronchus: Gangrenous Cavity: No Bronchiectasis.*

1906. Man, aged 69. Pain in the left chest with dyspnoea and foul sputum for six months. Note over the left chest was impaired, and there was dullness at the base; bronchial breathing and bronchophony; fatal haemoptysis.

Post Mortem.—The left bronchus at 2.5 c.cm. below the place of origin was infiltrated with a primary growth which extended to the bronchial glands. The bronchus opened direct into an irregular, foul, sloughing, gangrenous cavity for one and a half inches. The rest of the lower lobe was fibroid but no dilatation of bronchi was present; the upper lobe was becoming fibroid; the right lung was emphysematous; there was septic bronchopneumonia at the base.

CASE CXVI.—*Cancer of Left Bronchus: Bronchiectasis: Pleuritic Effusion.*

1900. Man, aged 36. Two years ago he had pleurisy, and again six months ago. There was blood-stained expectoration; the left chest was dull, with deficient breathing. On February 1st 36 oz. of fluid were aspirated; on February 26th 2 oz.; on March 12th 9 oz. of blood-stained fluid were withdrawn. There was increasing dyspnoea.

Post Mortem.—A carcinomatous growth had started in and occluded the left bronchus and invaded the lung; the tubes beyond were dilated and filled with pus. The pericardium was invaded by growth. The pleura was ⅛ in. thick; the lung was solid with growth, and there was bronchopneumonia.

CASE CXVII.—*Squamous Epithelioma infiltrating Wall of Trachea and Bronchi.*

1906. Woman, aged 17. Severe paroxysmal dyspnoea for one week; temperature 99.6; some muco-purulent expectoration; afterwards there was slightly deficient breathing at the left base. There was always a good vesicular murmur. Death resulted from dyspnoea.

Post Mortem.—There was diffuse infiltration, with apparently a primary growth of the wall of the trachea for a length of 6 c.cm. and for 3 c.cm. in each bronchus. The lumen of each tube partially reduced in size; there was no complete stenosis, no bronchopneumonia nor septic bronchiectasis in the lungs, but oedema was present; there was a trace of muco-pus in the tubes. The growth thickened the wall towards the lumen. Microscopically it was a squamous epithelioma, and no growth could be found elsewhere.